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Quotations on the Status of Dentistry

The greatly improved standing of the dental profession among the professions . . . . is one of the most striking changes in public opinion that I have witnessed during my seventy years of observation of educational progress. . . . . I do not think I have seen during my seventy years of observation of the professions and the means of training them any change so great as that which has taken place in regard to the dental profession, and to the means of training dentists.—*Charles W. Eliot, President Emeritus of Harvard University*, on "The progress of dentistry and of the dental profession." *Address before the Dental School Society of the Phillips Brooks House Association, Harvard University, December 8, 1924; Harvard Dental School, 1925, pp. 14 and 19.*

The dental school is one of the most important of these agencies [for the training of a qualified body of public health servants]. . . . . Today under the leadership of the best men in the profession it has set its face resolutely toward high service and honorable standards. . . . . It must have generous financial help, such as has been accorded in the last ten years to many of our medical schools, if it is to carry out its duty to the public health service. There is today no more direct method by which the public health can be served than to enable the universities to place their dental schools in a position to give the kind of education for which the world stands in need.—*Henry S. Pritchett, Ph.D., LL.D., Sc.D., Carnegie Foundation's Bulletin on Dental Education (1926); Preface, p. xx.*

I think it unlikely that physicians, unless they have a particular interest in the border subjects between medicine and dentistry, will maintain a permanent interest in dental research. This interest may be fostered for a period of special grants, but after these grants are used up or the individual matures, he will probably revert to medicine and not be a permanent investment for dentistry. . . . . The nucleus of . . . . [the] research group [for the advancement of dentistry] in my opinion must be dentists who have had the dental training and have a permanent interest in this field, who have taken the time to get additional training in some of the preclinical sciences. This of course corresponds to the make-up of the preclinical science group in medicine today. I feel a strong conviction, therefore, that individuals who would help dentistry should first help to develop research workers with dental training and dental interest.—*G. H. Whipple, A.B., M.D., M.A., Sc.D., Dean and Professor of Pathology, School of Medicine and Dentistry, University of Rochester; New York Journal of Dentistry*, 1931, i, p. 159; April.

The stomatologist's concept of dental education and practice finds expression in the activities of a very limited group in this country who, so far as I can see, are making no headway in dissecting a part of the practice of dentistry from its whole and grafting it on medicine; nor do I believe that medicine would welcome such implantation. I have no apprehension over the stomatologist's point of view. In the four countries in Europe, Austria, Czecho-Slovakia, Belgium and Italy, where one must possess a medical degree to practise dentistry, the poorest type of technical dental service is to be found. In Switzerland, Sweden, Norway and parts of Germany, where dentistry is taught in conjunction with medical studies, yet independent of medical supervision, as we teach it in this country, the best type of service is found. Based on my own study of the problem in Europe [1926–27], I would both seek and recommend the services of dentists trained in the latter countries during the past decade and would avoid service in the former, unless I knew the dentist and his work personally.—*Guy S. Millberry, D.D.S., Dean, Dental School, Univ. of Calif.; Pacific Dental Gazette, 1931, xxxix, p. 415; June.*

Nothing is to be gained by converting a first-class dentist into a mediocre medical practitioner. . . . . I do not believe it is practical to unify the dental and medical curricula so as to provide for the same training of dentists and physicians during any considerable part of their academic careers. . . . . Dentistry is a very specialized field of medicine which has already been elevated to a standard of professional excellence entirely comparable with that of other medical specialties. *The average American dentist is at least as well qualified to render adequate service in his field of work as is the average otolaryngologist, ophthalmologist, or surgeon* [italic not in the original]. Dental education in America has been notably effective, and that is to the credit of the profession. . . . . Changes should not be made at the expense of that which has been found good through long practical experience. . . . . I am . . . . entirely unconvinced that any plan for the unification of the dental and medical curricula would result in the

(*Continued on p. 3 of the cover*)

INTERNATIONAL ASSOCIATION FOR DENTAL RESEARCH

Scientific Proceedings of the Pittsburgh Section[1]
June 23, 1932

LAWRENCE E. VAN KIRK, B.A., D.D.S., M.S., F.A.C.D., *Editor*

The meeting was held in the auditorium of the Henry Clay Frick Training School, Thackeray Street near Fifth Avenue, Pittsburgh, Pa., June 23, 1932. *Chairman:* J. S. Ashbrook, D.D.S., F.A.C.D., Vice-president of the Section. The papers by Dr. H. E. Friesell, Dean of the University of Pittsburgh School of Dentistry, and Dr. J. J. Enright, a research specialist at the Mellon Institute, gave the first complete account of the results of the comprehensive scientific investigation of dental caries in progress at the University of Pittsburgh during the past nine years.

Scientific program

I. Studies of the cause and nature of dental caries. A. Introductory remarks, and clinical aspects of dental caries. *H. Edmund Friesell*, D.D.S., LL.D., Sc.D., F.A.C.D., Dean, School of Dentistry, University of Pittsburgh..... 570

II. Studies of the cause and nature of dental caries. B. Experimental investigation of dental caries. (a) Histological effects of acids on enamel. (b) Bacteriological studies of dental caries. *John J. Enright*, Ph.D., Senior Fellow, Mellon Institute..... 571

III. Discussion:

(A) *William J. Gies*, Ph.D., F.A.C.D., Professor of Biological Chemistry, Columbia University, New York City..... 572

(B) *Russell W. Bunting*, D.D.Sc., F.A.C.D., Professor of Oral Histology and Pathology, School of Dentistry, University of Michigan, Ann Arbor, Mich..... 573

(C) *Edward H. Hatton*, B.L., M.D., Professor of Pathology and Bacteriology, Northwestern University Dental School, Chicago, Ill..... 573

(D) *Major F. E. Rodriguez*, B.Sc., D.D.S., Dental Corps, U. S. Army, Camp Holabird, Md..... 573

[1] Devoted to researches on the nature and cause of dental caries. The papers and discussions will be published in an early issue of the *Journal of Dental Research*. For the last previous account of proceedings of the Pittsburgh Section see the *Journal of Dental Research*, 1932, xii, p. 117; February.

(E) *Philip Jay*, M.S., D.D.S., School of Dentistry, University of Michigan, Ann Arbor, Mich. 573
(F) *Theodor Rosebury*, D.D.S., Assistant Professor of Bacteriology, Columbia University, New York City 573
(G) *R. E. Morgan*, D.D.S., Pittsburgh, Pa. 574
(H) *Dr. Friesell* 574

Abstracts

I. Studies of the cause and nature of dental caries. A. Introductory remarks, and clinical aspects of dental caries

H. E. Friesell, D.D.S., LL.D., Sc.D., F.A.C.D., School of Dentistry, University of Pittsburgh, Pittsburgh, Pa.

Introduction. The etiologic and preventive aspects of dental caries are the major problems that confront the dental profession today. An increasingly large group of dentists believe that local environmental factors play only a minor rôle in the causation of tooth decay; that any program of prophylaxis based on oral hygiene is bound to be of slight value; and that the only hope of combating caries successfully is through diet. To study these problems a Multiple Fellowship was founded at the Mellon Institute in 1923. The Fellow has had the coöperation of many specialists at the University of Pittsburgh School of Dentistry in the fields of dentistry, histology, chemistry, bacteriology, and nutrition. An analysis of the literature indicated the need for a study designed to fill the gaps, and extend the boundaries of knowledge, in the concept concerning the active rather than the predisposing factors in the causation of decay of enamel. The author discussed recorded objections to Miller's theory, and some of the evidence tending to remove the objections; also the importance of proper nutrition during development of teeth; and concluded that deficiency in diet is influential primarily as a predisposing cause of enamel caries.

Some clinical aspects of dental caries. Certain outstanding clinical features of caries are apparently wholly ignored by various workers in the nutritional field. Dental caries is a chemical dissolution of the calcium salts of the teeth. It always begins on the surface of a tooth, never in the interior. It does not affect tooth surfaces uniformly, but begins at definite points, and is limited to specific areas. Areas

of tooth surface that are subjected to the scouring action of food, in mastication, are practically never attacked, unless developmental faults are present. Caries in such faults does not spread on the enamel surface. Fillings properly extended are preventive measures as well as reparative. Caries is rare on lingual surfaces, and is arrested on proximal surfaces under certain well-known environmental conditions. Decay does not begin on tooth areas that are kept clean. The peculiar reaction of carious enamel when submitted to the action of dilute lactic acid, and other acids, was discussed.

II. STUDIES OF THE CAUSE AND NATURE OF DENTAL CARIES. B. EXPERIMENTAL INVESTIGATION OF DENTAL CARIES. (A) HISTOLOGICAL EFFECTS OF ACIDS ON ENAMEL. (B) BACTERIOLOGICAL STUDIES OF DENTAL CARIES

J. J. Enright, Ph.D., Mellon Institute, Pittsburgh, Pa.

(a) *Histological effects of acids on enamel.* Effects on tooth enamel of its immersion in simple $\frac{\text{lactic acid}}{\text{sodium lactate}}$ and $\frac{\text{citric acid}}{\text{sodium citrate}}$ buffer-solutions that maintained relatively constant H-ion concentrations ranging from pH 4.0 to pH 8.0, for periods of from 1 to 60 days, were studied. Photomicrographs of ground longitudinal sections through the decalcified areas (after treatment of the teeth with silver nitrate) showed that (1) all of the simple buffer-solutions etched enamel appreciably, particularly in the longer immersion periods; (2) different teeth varied in their susceptibility to the action of the same solution; and (3) the appearance of certain ground sections of artificially produced caries closely resembled that of natural caries, in both early and advanced lesions. The positive effects in the neutral, slightly acid, or alkaline ranges could not be logically correlated with the observed durability of enamel bathed by saliva in the mouth. Evidence indicating that saliva is normally saturated with tri-calcium phosphate (calcium phosphates) was presented. Attempts were made to simulate in buffer solutions the conditions that prevail immediately in contact with a tooth undergoing carious degeneration. With the aid of buffer solutions that had previously been saturated with tri-calcium phosphate, it was found that the protection of tooth enamel against

the action of acid in the *saturated* buffers was marked in the pH-range from about 5.0 to 8.0, but that there was practically no protection in these buffers at pH 4.3 or pH 4.6.

(b) *Bacteriological studies of dental caries.* A bacteriological survey of progressive and also non-progressive caries, and of patients apparently "immune" to the disease, showed that the activity of the decay could be correlated with the presence of lactobacilli. This was particularly true when follow-up examinations of a bacteriological and clinical nature were made. The observed effects of acids on enamel, and the results of the bacteriological survey, confirmed the reports of many previous workers as to the importance of lactobacilli. Detailed studies of the characteristics of oral lactobacilli, as described by Bunting, Rodriguez, Morishita, and others, indicated that these organisms were identical with the type commonly found in the present work. Further comparison of oral strains with intestinal strains showed that the two main types of lactobacilli occur in the mouth and intestine, and that the smooth-colony type greatly predominates among the oral strains. A classification of relatively stable types of L. acidophilus was proposed, based on several correlated biochemical characteristics.

DISCUSSION

(A) *William J. Gies, Ph.D., F.A.C.D. (Columbia University, New York City)*: The (a) public, (b) dental profession, (c) Pittsburgh Section, (d) audience, and (e) Drs. Friesell and Enright were congratulated, respectively, on (a) the public benefits steadily accruing from the advancement of dental science and dental practice, (b) the earnest endeavors of the dental profession to meet its public responsibility by promoting this advancement, (c) the constructive activity of the Pittsburgh Section, (d) the opportunity of the large audience to receive the results of a prolonged and earnest research, (e) and the effective way in which a new solution of the problem of dental caries was sought and investigated. Like cancer and colds in the practice of medicine, caries in the practice of dentistry remains a curse and a puzzle requiring and receiving intensive attention for its solution. Current views on the origin and prevention of caries were given general attention not only to point out the complex difficulties Drs. Friesell and Enright have been obliged to face, but also to prepare the way for the more detailed and technical discussions by the succeeding speakers. Some of the physiological obstacles in such research were indicated.

(B) *Russell W. Bunting, D.D.Sc., F.A.C.D.* (*University of Michigan, Ann Arbor, Mich.*): The chemical and biological phases of dental caries, the influence of buffer substances in the saliva, bacterial overgrowths, and aciduric bacteria, were discussed in detail. "At the present time there is general concurrence in the view that dental caries is an infective process in which specific types of bacteria, acidogenic and aciduric in nature, are resident on the surface of a tooth and by their acid production destroy the tooth." Further conferences and coöperation, between such groups as are engaged in the study of this important disease, were strongly recommended.

(C) *Edward H. Hatton, B.L., M.D.* (*Northwestern University Dental School, Chicago, Ill.*): The importance of the clinical features of dental caries was emphasized. Any theory that does not explain all the known clinical facts about caries is unacceptable. Available data of research on the prevalence of dental caries during pregnancy refute the popular idea that pregnancy increases caries. Unjustified claims that nutritional factors determine the incidence of dental decay were pointed out and discussed.

(D) *Major F. E. Rodriguez, B.Sc., D.D.S.* (*Dental Corps, U. S. Army, Camp Holabird, Md.*): Dr. Rodriguez expressed pleasure for the opportunity to participate in this conference, which he believed would be most helpful in advancing our knowledge of dental caries. He strongly commended the decalcification experiments, and Dr. Enright's proposed classification of lactobacilli. The desirability of a quantitative rather than a qualitative technique, in making bacteriological surveys of the dental zone, was stressed.

(E) *Philip Jay, M.S., D.D.S.* (*University of Michigan, Ann Arbor, Mich.*): The papers were discussed from the bacteriologist's viewpoint. Research on methods for the control of overgrowths of B. acidophilus in the mouth was suggested. Manipulation of the diet has given the Michigan groups more encouraging results, in effectively diminishing such overgrowths, than any obtained with drug therapy. An immunologic approach was described. The bacteriologic findings in the Pittsburgh report were for the most part in accordance with those of the Michigan group.

(F) *Theodor Rosebury, D.D.S.* (*Columbia University, New York City*): Dr. Friesell's presentation of the clinical aspects of dental caries was commended as a much needed guide for research in this field. In the light of very recent experience it was suggested that experimental study of caries in animals promises to yield more significant results than any heretofore obtained in such work. The authors' classification of lactobacilli was approved as the best yet offered for stable or older strains. Freshly isolated strains might be expected to present greater heterogeneity. Dr. Rosebury's studies on the relationship between enamel solubility and the acidogenic

powers of lactobacilli, in terms of pH, have not thus far given him results similar to those of the authors. The problem is so complex and important that it warrants further study with various methods of approach.

(G) *R. E. Morgan, D.D.S. (Pittsburgh, Pa.)*: Attention was drawn to a patient in whom the saliva was markedly scant, this condition having been accompanied by rapidly progressing dental caries. The patient seemed to be healthy otherwise. The case was cited to point out the influence that deficiency in the quantity of saliva might have in accelerating the progress of caries.

(H) *Dr. Friesell (in conclusion)*: Speaking for the Pittsburgh Section, and for Dr. Enright and himself, Dr. Friesell indicated appreciation of the collaboration of those who participated in the discussion, and also of the sustained interest in the proceedings manifested by the large audience.

COMPARISON OF LOCAL EFFECTS OF ALPHA BUTYLOXYCINCHONINIC ACID-GAMMA DIETHYLETHYLENEDIAMIDE ("NUPERCAINE") WITH COCAINE AND PROCAINE

CHARLES DEW. DEYTON, D.D.S., M.D.S., JOHN W. WILSON, D.D.S., M.D.S., AND EARL A. ZAUS, S.B., M.D.

Department of Pharmacology, Northwestern University Dental School, Chicago, Ill.

Alpha butyloxycinchoninic acid-gamma diethylethylenediamide hydrochloride, first introduced as "Percaine," is one of many new synthetic local anesthetics developed during the last few years for which superiority over procaine is claimed. For topical use, Höfer (1), Lipschitz and Laubender (2), and Richter (3) have shown that it is more effective than cocaine. Kochmann (4) extends these findings to the field of infiltration, and recommends it as preferable to procaine, in spite of its greater toxicity as reported by Uhlmann (5), Bond and Bloom (6), Lake and Marshall (7), and Freund (8). Uhlmann (5) found "nupercaine" to be 10 times as effective as cocaine, 40 times as effective as procaine, and about 5 times as toxic as cocaine. Höfer (1) reported that subcutaneous injection was followed by prompt anesthesia lasting 6 to 12 hours, and observed that it is a greater vasodilator than procaine. Christ (9) showed that a 1–2000 solution with 1–100,000 adrenalin, injected subcutaneously, produced anesthesia lasting 10 hours and longer. Goldhahn (10) found that 1–2000 "nupercaine" solution produced anesthesia lasting 3 to 4 hours longer than 0.5 percent procaine solution. Pfefferkorn (11), using a series of 250 wheal tests as a basis of comparison, decided in favor of procaine. He reported greater burning at the site of injection, greater hyperemia, more marked late-infiltration, and anesthesia lasting but 15 minutes longer than that with procaine. Hackenbach (12), Christ (9), Höfer (1), Ritter (13), von Seeman (14), Goldhahn (10), and Keys and McLellan (15) reported, on the contrary, that "nupercaine" favors more rapid healing and is less irritating than procaine.

The observations in the dental clinics of Grabner (16) and Deveze (17) agree with the latter group.

The conflict in opinion among the many reports on "nupercaine" led us to investigate its local action, particularly with doses recommended for clinical use. We chose the rabbit cornea for topical measurements, and the wheal test for infiltration indices. We added a third test, recommended recently for determining anesthetic efficiency: the gold-fish method of Adams, et al. (18), as modified by Dailey and Benedict (19). While these tests were being carried out, the drug was tried in about 285 cases in our surgical clinic. (The results will be reported later.)

Materials and methods. Solutions.—In the rabbit-cornea tests, the dilutions were made with distilled water. In the wheal tests, Ringer solution was used because distilled water was too irritating to the skin in the control-wheal injections. These solutions were brought to 6.5 pH by adding 0.1 cc. of dilute hydrochloric acid to each 100 cc. Adrenalin, when used, was added just before the solutions were to be applied. A 1–500 "nupercaine" solution containing 1–16,000 adrenalin was added to the solutions to be tested, after it had been found in the surgical clinic that the other concentrations were inadequate for the production of good anesthesia and hemostasis. *Rabbit-cornea test.*—Solution was dropped into the conjunctival sac. Disappearance of pain sensation and the winking reflex were determined by touching the cornea with a stiff bristle. *Wheal test* [Braun (20)].—The radial surface of the forearm was the site of the injections. A tuberculin syringe (28-gauge needle) was used to inject 0.3 cc. of solution intracutaneously. A wheal about 0.5 inch in diameter developed. Control wheals were made with Ringer solution. The areas were tested for pain by pricking with a fine dental-explorer point. *Gold-fish test.*—Three- to four-gram gold fish were placed in about 250 cc. of solution at 25°C. Anesthesia was indicated when pressure upon the tail failed to elicit a response. The fish were then removed to distilled water to recover.

Results. Rabbit-cornea test.—In *tables 1* and *2*, "nupercaine" is seen to have been more effective than cocaine, procaine, and butyn. In clinical dosages (*table 2*), it had about the same onset-efficiency, while its duration efficiency was 2.8 times that of cocaine, 33 times

TABLE 1

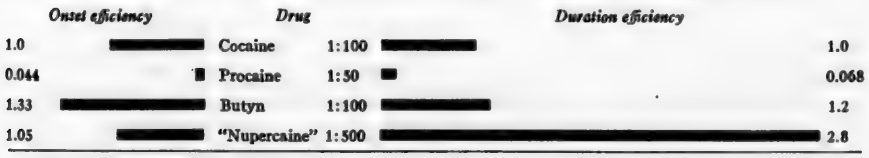
Rabbit-cornea tests

DRUG	CONCENTRATION (1 DROP)	ONSET TIME	DURATION	RATIO OF EFFICIENCY
Series A:				
Cocaine	1:50	27 sec.	44 min.	Unit
Procaine	1:50	15 min.	3 min.	0.068
Butyn	1:50	23 sec.	53 min.	1.2
"Nupercaine"	1:50	29 sec.	209 min.	4.7
Series B:				
Cocaine	1:100	40 sec.	39 min.	1.0
Procaine	1:100	0	0	—
Butyn	1:100	30 sec.	47 min.	1.2
"Nupercaine"	1:100	32 sec.	170 min.	4.3
Series C:				
Cocaine	1:500	54 sec.	16 min.	1.0
Butyn	1:500	48 sec.	26 min.	1.6
"Nupercaine"	1:500	38 sec.	110 min.	6.8

TABLE 2

*Rabbit-cornea tests**

DRUG	OPERATIVE DOSAGE (1 DROP)	TIME TO PRODUCE ANESTHESIA	DURATION
Cocaine	1:100	40 seconds	39 minutes
Procaine	1:50	15 minutes	3 minutes
Butyn	1:100	30 seconds	47 minutes
"Nupercaine"	1:500	38 seconds	110 minutes



* Scale: 0.5 inch = 1 unit.

that of procaine, and about 2.3 times that of butyn. A 1–500 solution produced anesthesia as rapidly as cocaine, and lasted about 1 hour longer. *Gold-fish test.*—Even with the weakest dilutions that induced "narcosis," the fish failed to recover following immersion in

the distilled water. Therefore, our results indicated death rather than narcosis, and cannot be used for comparison with the procaine and cocaine results. This is interesting in view of previous reports on this test [Benedict and Dailey (19)], which showed that it would accurately measure the anesthetic efficiency of local anesthetics of cocaine and procaine roots. Evidently, it cannot be used on the

TABLE 3

Wheal tests (without adrenalin)

DRUG	CONCENTRATION (0.3 CC. INJECTED)	QUALITY OF ANESTHESIA	DURATION OF ANESTHESIA	RATIO OF EFFICIENCY*
Series A:				
Cocaine	1:100	Complete	115 min.	1.0
Procaine	1:50	"	45 min.	0.38
"Nupercaine"	1:1,000	"	110 min.	0.96
"Nupercaine"	1:500	"	180 min.	1.56
Series B:				
Cocaine	1:2,500	"	9 min.	1.0
Procaine	1:2,500	"	5 min.	0.55
"Nupercaine"	1:50,000	"	25 min.	2.72
Series C:				
Cocaine	1:5,000	Incomplete	3 min.	1.0
Procaine	1:5,000	"	2 min.	0.66
"Nupercaine"	1:100,000	Complete	5 min.	1.6

* Scale: 0.5 inch = 1 unit.

quinoline roots. *Wheal test.*—In *tables 3* and *4*, comparisons are made with a wide range of dilutions. In the clinical range, 1-1000 "nupercaine" solution—adrenalized the same as the cocaine and procaine solutions—produced anesthesia lasting 40 minutes longer than with cocaine, and about 3 hours longer than with procaine. The 1–500 solution—adrenalized, 1–16,000—produced anesthesia lasting 85 minutes longer than with cocaine, and 3.75 hours longer than with procaine. These results confirm the clinical reports of Höfer (1) and Goldhahn (10). In our surgical clinic, increase in the

strength of the "nupercaine" solution from 1–1000 to 1–500 required an increase in adrenalization from 1–48,000 to 1–16,000 to control bleeding. A like change in concentration of the solutions in the wheal tests increased the anesthesia time but 45 minutes, indicating also the vasodilator effects of "nupercaine." Adding 1–48,000 adrenalin to the cocaine solution increased the anesthesia time 140

TABLE 4

Wheal tests (with adrenalin)

DRUG	CONCENTRATION (0.3 CC. INJECTED)	QUALITY OF ANESTHESIA	DURATION OF ANESTHESIA	RATIO OF EFFICIENCY*	
Series A:					
Adrenalin	1:48,000				
Cocaine	1:100	Complete	255 min.		1.0
Procaine	1:50	"	124 min.		0.48
"Nupercaine"	1:1,000	"	295 min.		1.15
"Nupercaine"	1:500	"	340 min.		1.33
Adrenalin	1:16,000				
Series B:					
Adrenalin	1:48,000				
Cocaine	1:2,500	Complete	44 min.		1.0
Procaine	1:2,500	"	36 min.		0.81
"Nupercaine"	1:50,000	"	53 min.		1.2
Series C:					
Adrenalin	1:48,000				
Cocaine	1:5,000	Incomplete	10 min.		1.0
Procaine	1:5,000	"	8 min.		0.8
"Nupercaine"	1:100,000	Complete	22 min.		2.20

* Scale: 0.5 inch = 1 unit.

minutes; adding 1–16,000 adrenalin to the 1–500 "nupercaine" solution increased anesthesia but 160 minutes.

Our observations did not agree with Pfefferkorn's (11) on the irritating effects of "nupercaine." Cocaine and procaine each produce greater irritation than "nupercaine," the reaction to the former resembling "histamine flare." Cocaine produced the most marked effects, and lasted about 10 days. Even with 1–500 "nupercaine" solution, the irritation was mild and disappeared completely within 48 hours, in agreement with the clinical reports already mentioned.

Summary. "Nupercaine" is more effective topically than cocaine or butyn.

"Nupercaine" is more effective for infiltration anesthesia than either cocaine or procaine, analgesia lasting three to four hours longer than with procaine. *Note.*—The toxicity of 1–500 solution would be an important factor in infiltrations requiring large amounts of the solution, but not to the dental surgeon who uses but 3–5 cc. of solution.

"Nupercaine" is less irritating to the tissues, and is apparently a greater vasodilator, than either cocaine or procaine.

The gold-fish test cannot be used as a test of the anesthetic efficiency of "nupercaine."

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SURFACE RESISTANCE OF HUMAN ENAMEL TO ACID DECALCIFICATION

EDWARD C. DOBBS, D.D.S.[1]

Department of Biochemistry and Pharmacology, School of Medicine and Dentistry, University of Rochester, Rochester, N. Y.

Introduction. The enamel surface has greater physical resistance than the sub-surface. Karlström (1) found that the hardness of human enamel increases from the dento-enamel junction outward, and that there is marked increase in hardness at the enamel surface. The Herbert Pendulum Hardness Tester was used for his determinations. He found that increase in hardness was associated with increase in specific gravity, and concluded that, as between erupted and unerupted teeth, there is no appreciable difference in hardness in the outer 20 percent of the enamel. Bierbaum and Hodge (2), using a Microcharacter, likewise found enamel surfaces harder than sub-surface areas. In extending these investigations on the *physical* properties of enamel, I have attempted to correlate the physical and chemical properties by determining the resistance of enamel surfaces and subsurfaces to the action of acid.

Experimental. Extracted human teeth, when immersed in weak solutions of organic acids, were decalcified first on the worn enamel-surfaces.[2] These areas were sufficiently abraded to expose the subsurface to the acid. The areas that were not decalcified by the acid had been protected by existing dental plaques, which could be floated off by immersing the teeth in 5 percent HCl. Teeth "deplaqued" in this way were white—and clean, excepting where areas of calculus had not been removed by the treatment. Such "deplaqued" teeth, when immersed in 0.75 percent lactic acid, were decalcified uniformly on the unworn enamel surfaces. The surface-resisting property of enamel

[1] Rockefeller Fellow in Dentistry.

[2] Decalcification of the enamel was determined by scratching the tooth with a sharp-pointed instrument. Teeth showing softening were classified as decalcified. This softening is always associated with whitening of the enamel.

against the action of dilute lactic acid is evidently removed with the dental plaque. When teeth were cleaned with a motor-driven brush and pumice, this surface-resisting property was not removed. Therefore this surface protection is not derived from the collection of material that adheres loosely to enamel. When teeth similarly cleaned, and from which small areas of the surface enamel had been removed by grinding with a carborundum stone and polished with a finer stone, were immersed in an acid solution (0.75 percent lactic), decalcification occurred on the ground areas and on the areas worn by mastication, but not on the unworn enamel surfaces, thereby demonstrating that the unworn enamel surface is not so susceptible to acid decalcification as the enamel sub-surface.

Teeth cleaned by immersion in alkaline trypsin-solution for three days, at 37°C., did not show the surface-protecting property. There were a few exceptions. Thus, a small area on a protected surface or in a depression might not decalcify, but the adjacent surfaces would. Choquet found Nasmyth's membrane present on only 12 percent of adult erupted teeth. This fact suggests that these "small areas" that did not decalcify were protected by remains of this keratinous membrane, which had not been removed by attrition. A thin colorless membrane was detached from these protected spots with HCl, suggesting that a membrane was the protecting material. Tryptic digestion removed the mucin plaques, but not the keratin membranes (3), demonstrating that the surface protection was not due to the keratin fragments but to the acquired mucin plaques that adhered *firmly* to the enamel surfaces.

Teeth that had been cleaned by brushing, and had shown this surface-protecting property, were studied under a microscope with vertical illumination. Transitional areas adjacent to worn decalcified surfaces showed evidence of decalcification on the crests of the imbrication ridges (4), and absence of decalcification in the grooves between the ridges. This condition, also present on teeth cleaned with trypsin solution, represents a keratinized protein in the depressions where attrition had not removed it.

The dental plaques that were removed from the enamel surface with HCl in an earlier experiment were tested for the presence of indigestible protein. By digesting the plaques for three days in pepsin

—HCl solution at 37°C., washing in 5 percent NaOH for a day, extracting fats with petroleum ether, and then digesting with trypsin for three days at 37°C., undigested organic material remained. The HCl removed from the enamel surface the acquired mucin plaques and the fragments of the keratin membrane. The tests on teeth cleaned with alkaline trypsin-solution showed that this keratin membrane does not appreciably protect the enamel surface against the action of acids. The slight amount of this indigestible protein in a plaque suggests that it is present on the enamel surface of adult teeth in very small quantity. This agrees with the results of an earlier experiment mentioned above, where membranes were found only on small isolated spots and in the imbrication grooves of some of the teeth. The indigestible part of dental plaque had the properties indicated by the following findings: Xanthoproteic test, positive. Biuret test, positive. Na_2S produced no swelling. Concentrated H_2SO_4, HCl, HNO_3, and KOH did not dissolve it. Hot 15 percent NaOH, applied for two hours, did not disorganize the mass. This indigestible part of plaque is evidently very resistant to acids and hydroxides, does not swell in Na_2S, and gives positive protein tests. It appears that this material is keratin, resembling neurokeratin more than epidermal keratin (hair or nails), which swells in Na_2S (3).

Teeth cleaned with trypsin solution were divided into two groups. From the first, the enamel surface was superficially removed. On the second, the deeper layers of the enamel were exposed. When these teeth were immersed in weak lactic acid solution, the teeth on which the deeper layers of enamel had been exposed were decalcified first; the superficial layers, later. In the light of Karlström's results (1)—the deeper layers of the enamel are softer and have a lower specific gravity than the enamel surface. We may conclude, from the above findings, that the physical properties and chemical properties, as indicated by acid decalcification, are associated, i.e., the greater the hardness and specific gravity, the greater the resistance of enamel to acid decalcification.

This surface phenomenon was investigated also on unerupted teeth. A small area of the enamel surface was removed with a stone, and the teeth immersed in 0.75 percent lactic acid solution. There was no surface protection; all exposed areas of the enamel decalcified uni-

formly. These teeth were put in 5 percent HCl; a thin colorless membrane floated away from the enamel surface of some of them. When this membrane was stained with methylene blue, two types of tissue were seen: (*a*) a clear, undifferentiated film, showing impressions resembling enamel-rod faces and imbrication lines; and (*b*) a cellular tissue that took a deep stain. Under the microscope, this membrane differs from the protecting membrane on erupted teeth, the former having a more definite pattern and cellular structure; the latter being an undifferentiated, deeply-stained mass.

The enamel surface of fully developed unerupted molar teeth did not show the surface protection to acid decalcification that erupted teeth had exhibited. This was due not only to the absence of the mucin plaque and the fully keratinized enamel membrane, but also to the lack of resistance of the enamel. There was no appreciable difference in the rates of decalcification between the enamel surface and the sub-surface in unerupted teeth, although such a difference was noted for erupted teeth. Enamel of unerupted teeth is evidently changed after eruption, becoming more resistant to acid decalcification.

Conclusions. Extracted teeth, immersed in 0.75 percent lactic acid, are decalcified first on the clean worn-surfaces. Cleaning the teeth with pumice and a motor-driven brush does not remove the surface-protecting property of enamel against acid decalcification. Tryptic digestion removes the surface-resisting property of enamel, making the unworn surfaces *uniformly* susceptible to acid decalcification. Unerupted teeth do not have the surface-resisting property shown by erupted teeth. The protective membranes on unworn enamel surfaces are organic in nature, and acquired after the tooth erupts. The deeper layers of enamel are more susceptible to acid decalcification than the enamel surface. There is an association between the physical properties of human enamel and the rate of acid decalcification.

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THE CONTROL OF DENTISTRY BY THE MEDICAL PROFESSION[1]

HUBERT C. VISICK, L.D.S., R.C.S. ENG.

"The control of dentistry by the medical profession" has been a burning question with me ever since my student days. After my paper was written, I was astonished to learn from the presidential address of Mr. Kittow that some eighty years ago this very matter was the subject of long and heated dispute between those who held that the profession of dentistry should be amalgamated with that of medicine, and those who desired that we should remain a distinct and separate entity, fearing that otherwise we should become "an excrescence on the tail of the College of Surgeons." Those in favour of amalgamation won, and now to-day we are able to review the position in the light of experience. In my opinion the fears of those who wished to keep dentistry as a profession apart were fully justified.

The Tree of Healing has many branches—medicine, surgery, dentistry, midwifery, massage, radiography, etc. All grow from the parent stem, some of the branches being bigger and more important than others. Emphatically, dentistry is a branch and not a mere twig growing on that of medicine, and I contend that the attempt to make it so is responsible for the miserably low standard of dentistry in this country. The dental profession is completely lacking in self-reliance after all these years of subservience and dependence, and is like a flock of bewildered sheep, harassed on every side by conflicting interests. I trace this condition of things to the fact that we are dominated and controlled by people who know nothing about dentistry—the General Medical Council.

Comparisons are supposed to be odious, but let us face the facts.

[1] Abstract of the presidential address delivered at the annual meeting of the Southern Counties Branch of the British Dental Association, Bognor, October 17, 1931. Reprinted, by permission of the author and by courtesy of the editor, from the *British Dental Journal*, liii, pp. 698–702; June 1, 1932.

America the world over holds the premier position in dentistry. We in this country seem to be quite content to follow in her train—making use of her ideas and her appliances. Can you think of a single article of modern surgery equipment which did not originate in the States, and where do you have to go if you want the very best? Compare the work in the mouths of our patients with the best which we know can be done, and I think we shall all admit that we—both patients and dentists—are content with very poor stuff indeed. The hosts of slovenly amalgam fillings in badly prepared cavities, the hideous prosthetic work disfiguring the faces of our patients—here is neither art nor science. Scaling, the foundation of proper dental treatment and probably the most important of all dental operations for the preservation of the health of the mouth, appears to be almost unknown in this country. Orthodontics might be called an American specialty, and we turn to them for practically all our ideas of this work. At the recent International Orthodontic Congress in London, out of thirty-three demonstrations only five were British.

It needs considerable courage to face the facts when education is considered. After I qualified at Guy's in 1901, I visited twenty-eight schools in America where I spent two years, and on my return to this country I contended that we were twenty years behind the time; if I had said thirty years it would have been nearer the truth. I think an explanation may be found in the teaching. American teachers make dental instruction their life's work, while with our instructors it has been only a side line. Each branch of dentistry in America is taught by men specialising in that branch. The whole atmosphere of the school is an inspiration, and if a young man wants to improve his dentistry, six months in America will do him more good and make him a better dentist than taking half a dozen medical degrees. Why is it that in dental affairs England is so far behind America? It seems to me that the reason lies in the control of dentistry by the medical profession. We have to thank that alien control for: (1) Our inadequate education, overloaded with useless subjects at the expense of essentials, such as mechanics and manual dexterity; (2) the swamping of our profession in the past by hordes of unqualified men, a direct result of the neglect and blindness of the General Medical Council; (3) the depressing psychological effect of the association, upon unequal

terms, with the medical profession, and the consequent low esteem in which our profession is held.

We have become so accustomed to this subservient position that we do not realise what freedom from outside control would mean. If any of you have been to the States you will have noted the tremendous enthusiasm, keenness, and *esprit de corps* of the dentists there. They are proud of the fact that they are dentists, and believe wholeheartedly in the importance of their profession. Their meetings, attended by thousands, have an inspiring effect. I do not think it is an exaggeration to say that there are hundreds of demonstrations at a single meeting. In the States, dentistry is, and always has been, entirely free from any outside control. No medical man is allowed to practise dentistry. The curriculum is arranged, the subjects are taught and the students are examined by dentists alone. Doesn't this freedom of the American profession fill you with envy? Is it any wonder they have made such a success of dentistry? . . . .

It was an evil moment when in 1878 dentistry in England was placed under the control of the General Medical Council. Naturally, our predecessors were proud to be associated with such a noble body of men, but would they have welcomed this change if they had realised that instead of being received on terms of equality, they were entering a veritable Egyptian bondage? Did they realise that the Act gave them not one single representative on the Council—this body which was in future to have absolute control of the education and registration of dentists? Had they foreseen this humiliation, they would have spurned the alliance. For forty-three years, we have the astounding spectacle of the control of the education of the members of our beloved profession being in the hands of men who knew nothing whatever about dentistry. And not only were they ignorant of it, but they did not conceal their contempt for it, and scornfully referred to dentists as mere "tooth-pullers." Do you realise that to-day the Council consists of thirty-eight medical men and not a single dentist? We are merely represented by three dentists from the Dental Board, who are co-opted when dental matters are under discussion? Naturally when such a body was asked to arrange the education of "tooth-pullers" they looked at it from their own point of view, and said that if such men were to be associated with them, their simple and empirical

knowledge must be rounded out with a padding of medical and surgical study to make them respectable members of the fraternity. They could not realise that our work touches medicine and surgery at certain points only, and to a very limited extent. Dentistry is essentially a handicraft. The dentist, surrounded with complicated machinery, spends his life with tools in his hand, wrestling with mechanical problems demanding almost superhuman manual dexterity. Operations in the mouth require sureness of eye, steadiness of hand, rapidity of movement, and on occasion, great strength scientifically controlled. The dentist must be master of his materials —gold, silver, porcelain, amalgam, the plastic denture bases and hosts of other substances, each demanding a special technique, but this aspect of dentistry can never be fully revealed to anyone but a dentist.

The General Medical Council formulated what it considered a suitable dental curriculum and laboured to produce a sort of medico-dental hybrid with too much emphasis on the tooth-pulling aspect of our work. These graduates, overloaded with theory and non-dental knowledge, were both unsuited by their education and insufficient in numbers to meet the needs of the country. The masses were crying out for plain dentists, practical men who could relieve them of their aching teeth and provide them with substitutes. Unqualified men began supplying this demand. There were many shameless humbugs among them, but the founders and backbone of this movement were mostly dental mechanics thoroughly trained in the one essential, manual dexterity. The General Medical Council refused to see the danger and, in 1921, forty-three years after they had taken control of dentistry, our profession, almost submerged beneath a sea of unqualified men, had to pay the price of their incompetence. The reason why these unqualified men, who possessed none of the knowledge deemed requisite by the Council, looked like beating us at our own game was because they possessed the one essential—manual dexterity. Although we seem ashamed to admit it, dentistry, in its simplest form, is mechanics and nothing more.

The Act of 1921 gave us a Dental Board of our own. But stop and look at its composition—four laymen, three medicals and six dentists! In other words, the dentists on the Dental Board are in the minority. If this Board confined its activities to holding the balance between dentists and the public, it would not be so humiliating, but this body

is dabbling in matters exclusively dental, spending money received from dentists on bursaries, grants in aid of research, dental education and poster propaganda. Thus a body, the majority of whom are not dentists, in control of our money, are having the audacity to dictate which subjects are the most important for dental research and education. Here is the Dental Board gaily decorating the outside of the house in an attempt to camouflage its defects; but, for those who have eyes to see, these very activities are convincing evidence of the rottenness of the structure. To adopt a railroad simile, dentistry has got on to the wrong line. Instead of a dental train controlled by a dental staff, running on its own lines, the dental profession is relegated to a van at the tail-end of the medical train, with practically no choice as to its destination. True, in recent years the dental van has been put in the charge of a conductor, who acts as a spokesman and is the virtual figure-head of the profession, but it is mortifying to think that this conductor is not a dentist, not even a doctor, but just a layman, someone who knows nothing whatever about the teeth. The Chairman of the Dental Board is an exceedingly kind and tactful gentleman, who makes us feel our position as little as possible, but the fact remains that we are submitting to something to which no other profession would submit for a moment. It may be argued that the conditions are peculiar, but would the Church, the Law or Medicine submit to like conditions?

The psychological effect of this domination by the medical profession is, that the atmosphere surrounding our education has led us to accept the suggestion that our occupation is an inferior one, and that it needs outside support. It is a common thing for a dental student to be urged to take a medical degree, the argument being, not that this will make him a better dentist, but that it will give him a better standing. Imagine what the effect must be on a self-respecting young man on finding that the profession he has chosen for his life's work is held in such low esteem. The right type of educated man cannot be expected to take up dentistry while such a state of affairs exists. How often do dentists who rise to positions of eminence quietly jettison their dental qualification. Medical and surgical degrees are admitted, but the L.D.S. is missing. In one of the London Dental Schools the dentist in charge is called the *Medical* Superintendent. Could absurdity and confusion be carried further?

I must not conclude without putting forward constructive proposals. First of all we must have a united profession. I was one of the die-hards who opposed the registration of unqualified men, but to-day they are, for better, for worse, on the Register, and our fellow practitioners. I am convinced that for the sake of dentistry we must unite. United we may succeed, divided we are bound to fail. The next step is self-government. The only hope for dentistry is to make a clean break with the traditions tying us to the apron-strings of the medical profession. I should like to see a rebellion, the dentists rising in a body and saying that they wlll no longer tolerate the ignominy of their position, that they intend to run their own train with only dentists to control it. The education and examination of dentists must be entirely in the hands of dentists. The problem we have to face is, that there are two grades of dentistry needed. First of all, a very simple form of dental treatment is required for the great majority of our population; given intelligent organisation it should be possible to shorten the course and still to turn out a dentist capable of supplying the needs of the masses, but we must have a more advanced course for those who have the means and ambition for longer study. Every dental school must have a dentist dean, an experienced educationist in daily attendance. This is essential, for he must keep in close touch with the students and see that not a moment is wasted. He must have authority to organise and control every bit of the teaching. I will say no more except to express the hope that at the end of their studies our students would be examined by the Royal College of Dentists.

May I conclude with a confession of my faith in dentistry? I am the son of a dentist, taught from my earliest years to reverence dentistry. My conviction after thirty years' experience is that dentistry is worthy of any man's highest endeavour. However clever, dexterous or artistic a man may be, he can exhaust every ounce of his talent in the ordinary everyday work of a dentist. I believe that if we desire to live a useful, happy life of service for our fellow men, we can find in dentistry full scope for all our aspirations—

"Room to deny ourselves, a road
To bring us daily nearer God."

The Wolds, College Road,
Eastbourne, England.

AN ENDEMIC CENTER OF MOTTLED ENAMEL IN CHINA[1]

BERT G. ANDERSON, D.D.S.

Department of Surgery, School of Medicine, Yale University, New Haven, Conn.

The occurrence of mottled enamel in China was first reported by Anderson and Stevenson in 1930 (1). This report dealt primarily with the more general distribution of the disease throughout North China, although mention was made of the existence of endemic centers in some regions. These investigators studied the incidence of the abnormality, the severity of the condition, and the two phases of the enamel defect; namely, the chalky and the brownin-stain features. The present report deals with the investigation of an endemic center in the province of Ho Pei. The finding of a marked case of mottled enamel in an individual from Lung Feng Chuang suggested a special investigation of the condition of a random group of the native population of his village. Lung Feng Chuang is a small village in the Province of Ho Pei, about 20 miles south of a point on the railway midway between Tientsin and Peiping. A day's overland travel in a "Peking Cart," after leaving the railway, brings one to this secluded village. Its population of about 250 eke out their existence mainly by means of agriculture. The diet of the people of this region was studied by Adolph in 1929 (2). Open wells, like those commonly seen throughout China, furnish the water supply.

All who were willing to have their teeth inspected were examined for mottled enamel. The number examined was fifty-four, or approximately 20 percent of the population. The examination of small groups was conducted in the open streets wherever a few individuals were available. The majority of those examined were males ranging in age from youth to late adulthood. No record was made of age or sex. In each case the presence or absence of both chalky and brownin-

[1] Read at a meeting of the New Haven Section of the International Association for Dental Research, Dec. 7, 1931. The material for this paper was collected while the author was a member of the staff of the Peiping Union Medical College, Peiping, China.

stained enamel was noted. The amount of chalkiness or brownin stain in each case was estimated and recorded as degree 1, 2, 3, or 4. A slight degree of occurrence was represented by 1, while 2, 3, and 4 consecutively, represented increasing degrees of severity of either condition. The complete absence of either phase of the disease was indicated by 0. The photographic guide used as a standard in estimating the degree of defect present in each case was shown on page 235 of the paper by Anderson and Stevenson (1). The investigation yielded the results assembled in the accompanying summary.

Summary: Showing (*A*) occurrence of different degrees of chalky enamel; and (*B*) occurrence of different degrees of brownin-stained enamel

(*A*)	*Normal*		*Chalky*			*Total number examined*
Degree of defect.....	0	1	2	3	4	
Number having defect..	0	2	9	21	22	54

(*B*)	*Normal*	*Brownin stained*				*Total number examined*
Degree of defect.....	0	1	2	3	4	
Number having defect..	0	12	15	21	6	54

The data show that mottled enamel was present in all the individuals examined. The random group that was systematically examined probably represents a fair cross-section of the whole population of the village. The condition was present also in all individuals whose teeth were only casually seen. Besides being more widespread in its distribution among these people, the condition was found to be far more severe than in either of the two groups previously reported by Anderson and Stevenson (1). A comparison of section *A* with section *B* in the summary shows that the chalky feature of this enamel defect was considerably more aggravated than the discoloration. One would expect this to be so since it has been recognized that the staining is secondary to the faulty enamel structure (3).

Many of the endemic centers of mottled enamel described by McKay (4) as occurring in Europe and America are found in regions where artesian water is used for drinking purposes. The frequent association of artesian water with this enamel defect led McKay to conclude that *artesian* water was directly related to the causation of mottled

enamel. The water supply of Lung Feng Chuang, where the people have marked and widespread mottled enamel, comes from shallow open wells. Smith, Lantz, and Smith (5) have found that the water supply of endemic mottled-enamel centers in Arizona contains abnormal amounts of fluorine. Furthermore, they claim to have produced this enamel defect experimentally, both by giving rats drinking water taken from wells in these endemic centers and by incorporating, into the diets of rats, the residue from the evaporated water. Churchill (6) has also found that water from endemic centers of mottled enamel contains unsuspected amounts of fluorine. He does not feel that a causal relationship is implied because of the association of mottled enamel with excessive amounts of fluoride in the water. The writer is attempting to have samples of the water from the wells of Lung Feng Chuang analyzed, with special reference to the fluorine content. The results of this analysis will be published when the data become available.

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APICOSTOMY: A METHOD TO OBTAIN CULTURES FROM PERIAPICAL TISSUE*

LOUIS I. GROSSMAN, D.D.S., DR. MED. DENT. (*Rostock*)
School of Dentistry, University of Pennsylvania, Philadelphia, Pa.

Introduction. *Apicostomy* is a descriptive term applied to an operation in which a sterile channel is created to obtain cultures from the apical end of a tooth, by cutting through the overlying soft tissue and bone. It is derived from L. *apex* (apicis = end of a thing), L. *os.* (ossis = bone), and G. *tomē* (= incision). The instrument for the attainment of this objective is called *apicostome.* It consists essentially of a trocar and cannula suitably held in the handpiece of a dental engine, by which it is revolved. The purpose of apicostomy is to provide a sterile avenue or channel of approach to the apical tissue to obtain uncontaminated cultures, the latter being used to determine the bacteriologic condition of this tissue. In its clinical application the method can be used (*a*) to determine the status of a tooth suspected of being a focus of infection; (*b*) to determine whether sterility of the apical tissue, once obtained, can be maintained over an extended period of time; (*c*) to evaluate a method, or different methods, of root-canal therapy by bacteriologic control.

In 1918 Coriell[1] reported the use of a specially devised instrument to obtain cultures from root apices under aseptic conditions. This consisted of a cannula, attached to the contra-angle handpiece of the dental engine, into which fitted a trocar or twist drill. By revolving both the trocar and cannula, the gum and bone tissues could be pierced and the tooth apex reached. The trocar being then removed, and the cannula left in place, a direct approach to the apical region was thereby provided. The operation, of course, was done under a suit-

* This work has been aided by a grant from the Research Fund of the Academy of Stomatology, Philadelphia, Pa.

[1] Coriell, L. D.: A dental trocar, *Dent. Cosmos*, 1918, *60*, 1154; Dec.

able anesthetic and with due regard for asepsis. In 1931, Grossman[2] attempted to determine the frequency with which the apical region is reached by this method. It is evident that were the instrument misdirected in its path and the apical region not attained, a false cultural report would result. The use of the method would therefore not justify itself. In an anatomic study performed on cadavers he found that in 49 apicostomies, the apical region was reached in about

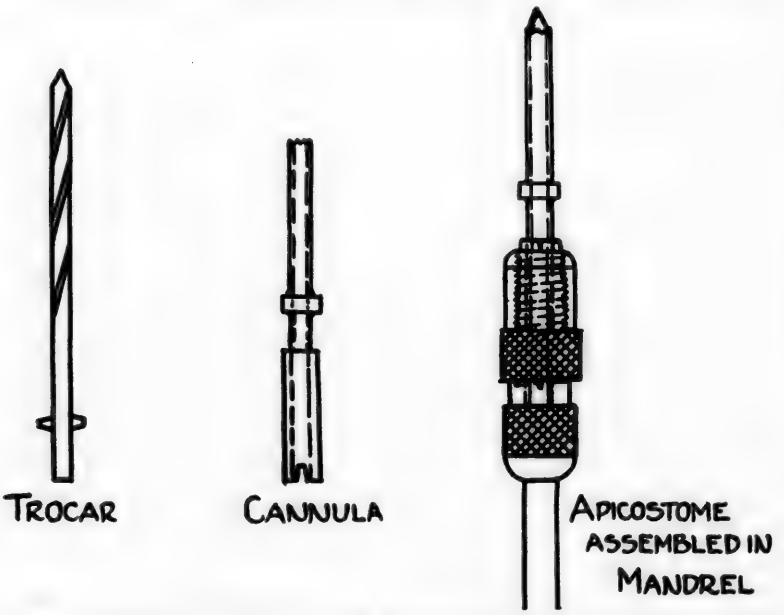

FIG. 1. Apicostome: trocar and cannula shown separately, and assembled in mandrel.

40, or 80 percent of the cases. It was concluded, from this study, that the number of successful attempts justified the use of the method. In the course of the work, the ends of two trocars were broken off and lodged in the tissues. It was also felt that the direction of the apicostome could be guided better toward the apical region, if the instrument were attached to the straight, rather than the contra-angle, handpiece. The design and construction of the Coriell apicostome,

[2] Grossman, L. I.: Bacteriologic control of periapical tissue by the Coriell trocar method, *Dent. Cosmos*, 1931, *73*, 219; March.

however, made its use in the straight handpiece impracticable. These observations, together with the fact that the contra-angle handpiece does not lend itself readily to sterilization, led to the development of the apicostome illustrated here (*figs. 1–3*).[3]

Apicostome. The design of this instrument allows the use of a thicker, slightly heavier trocar, thereby reducing the possibility of

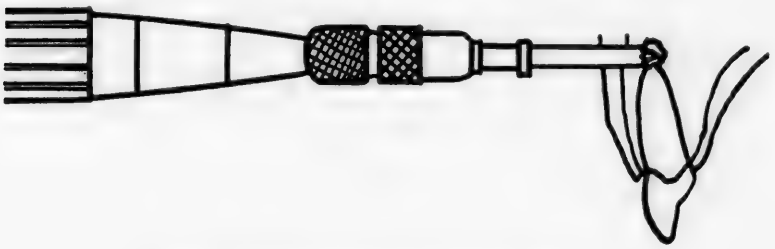

FIG. 2. Apicostome directed to apex of tooth.

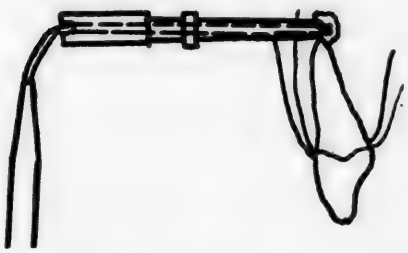

FIG. 3. Cannula in place, trocar removed; ready for insertion of platinum wire for removal of apical material to culture medium.

breakage. When assembled, the trocar and cannula fit into an ordinary dental mandrel, which in turn is screwed into the straight handpiece. This type of apicostome has an additional advantage: The three parts—cannula, trocar, mandrel—are assembled before sterilization and, after being sterilized, are attached to the handpiece as a unit, thus lessening the danger of accidental contamination (*fig. 1*). Al-

[3] This instrument has been made by Mr. John Burkhardt, 263 South Eleventh St., Philadelphia, Pa.

though the trocar and cannula are made of high-carbon steel, which minimizes the likelihood of breakage even after prolonged use, it is a good rule never to use the instrument for more than 10 or 12 operations. By this time the serrated edges of the cannula as well as the cutting blades of the trocar become dulled, and the instrument begins to show other signs of deterioration that warrant its rejection. It is more judicious and economical to replace the instrument than to incur the risk of the injury to a patient, and perhaps of the lawsuit, that might result from breakage of the trocar in the alveolar bone. The fact that the author has not experienced an accident of this nature in more than 30 apicostomies is attributed to the extreme caution exercised in this respect.

Apicostomy must be looked upon as a surgical operation requiring a strict aseptic technic. It is not sufficient that the periapical tissue be found in an aseptic condition—it must be left that way. Only the six anterior teeth, both upper and lower, and occasionally uniradicular premolar teeth, lend themselves to this operation. The method is not applicable to multirooted teeth. In operating on lower teeth it is sometimes desirable to prepare the patient with atropin, to lessen the salivary secretion.

Apicostomy. The operation may be performed under either general or local anesthesia. The author prefers local anesthesia. Where gross apical infection is suspected, block anesthesia should be employed. Ordinarily, infiltration of the tissues with 2 cc. of 1.5 percent novocain solution produces sufficient anesthesia. After the tissues are anesthetized the remainder of the instruments, cultures, etc., are put in readiness. The handpiece is sterilized by repeated washing with alcohol, or one of the new "boilable" handpieces may be used. The freshly sterilized apicostome already assembled in the mandrel is then attached to the handpiece. The field of operation is swabbed with 1.25 percent iodin in isopropyl alcohol and decolorized with 90 percent alcohol. Sterile cotton-rolls are used to keep the field dry and uncontaminated. A control culture is taken at this stage. The apicostome is now directed at the apex of the tooth. The point of entrance of the apicostome is previously determined by measuring the length of the tooth on a roentgenogram, from incisal edge to apex. Care must be exercised in directing the apicostome at a suitable angle

so that the apical region will be reached (*fig. 2*). As in roentgenology, where improper angulation of x-rays produces either foreshortening or elongation of the object, so here—the apicostome may either strike short of the apical region or go far beyond it. Here, as in other work, sufficient practice affords the necessary experience.

When the apical region is attained, the mandrel is unscrewed and disengaged from the apicostome. Sterile-cotton pliers are now placed against the hub of the cannula, to hold it in position, while the trocar is withdrawn and a culture taken from the osseous material clinging to the end of the trocar; or the trocar itself containing this material may be dropped into a tube of culture medium. A sterile platinum needle, the end of which has previously been flattened, is now inserted

FIG. 4

FIG. 5

FIG. 4. Radiopaque image produced by 20 percent sodium iodid in vaseline.

FIG. 5. Radiopaque image produced by sodium iodid in wax paste.

into the cannula, rotated a few times to scoop out material, and used to inoculate suitable culture media (*fig. 3*). The cannula is then withdrawn, the wound sterilized with a suitable antiseptic, and covered with a cotton roll for several minutes. A roentgenogram should be taken at this stage to determine whether the apicostome attained the apical region. For this purpose, Lipiodol, a radiopaque iodized oil, may be injected into the wound through a hypodermic syringe. While this agent is usually satisfactory, occasionally some of the oil flows out of the wound, covers the adjacent tissue, and produces a smudge-like image on the roentgenogram. Recently, a bone paste composed of vaselin (80 parts) and levigated sodium iodid (20 parts) has been injected into the wound following apicostomy. This pro-

vides a sterile plug that prevents infection. By virtue of the radiopacity of the paste, the exact location of the wound in relation to the tooth apex can be determined roentgenologically (*fig. 4*). The paste may be injected into the wound directly through the cannula, or through the opening created by the apicostome after it is removed. For this purpose a hypodermic syringe may be used to which a No. 20- or No. 21-gauge needle is attached. Should a heavier-bodied paste be found more desirable, the following mixture, in the parts indicated, may be used: Yellow wax, 15; vaselin, 15; oil of almond, 25; levigated sodium iodid, 12; M.F. pasta. This is more in the nature of a radiopaque bone-wax, and should be warmed slightly over a water bath just before use (*fig. 5*).

Following apicostomy the wound heals rapidly leaving no visible trace of the operation. The bone paste readily disappears. Swelling and pain rarely occur. Should they develop, the ordinary remedial measures should be instituted.

Summary. A method by which cultures may be obtained from periapical tissue is described. An improved type of apicostome is presented. The technic of apicostomy is given.

OCCURRENCE OF ENAMEL TUFTS[1]

THEO. B. BEUST, M.D., D.D.S., F.A.C.D.

School of Dentistry, University of Louisville, Louisville, Ky.

The universal occurrence and regular distribution of the enamel tufts awaken the belief that they are important or necessary constituents of human enamel (*fig. 1*). Their significance has been a matter of considerable speculation. In 1912 I showed (1) that tufts are permeated by stains injected into the pulp cavity, which proves their connection with the circulatory mechanism of the pulp. For many years I have regarded them as avenues of interchange for tissue fluid coursing between the dentin and enamel. I am aware that some doubt the occurrence of such an interchange, many in fact even denying the claim that the tufts seen in ground sections can be stained. Because of these contrary views I present these natural-color photomicrographs, showing the effects of staining through the pulp cavity. (Demonstration of photomicrographs in natural colors.) These photographs furnish indisputable proof that the enamel is connected with the pulp by open channels. These specimens were made from teeth which were first stained and afterward sectioned.

We must conclude from these and other observations that the tufts are anatomical structures definitely correlated with the occurrence of metabolic changes in the enamel. As this view is not universally held, some prefer to regard the tufts as defects or artefacts produced during the preparation of the specimen. These opinions have led to examinations into the effects of traumatism as a factor in tuft formation. This phase of the investigation was considered in a summary of views on enamel metabolism recently compiled by Chase (6), who cites (p. 700) four authors who claim that tufts are cracks. On the same page we read Chase's statement that "Akamatsu's contention,

[1] Work supported, in part, by a grant from the American Dental Association. Read at the Tenth General Meeting of the International Association for Dental Research, Columbus, O., March 19 and 20, 1932; *Journal of Dental Research*, 1932, xii, p. 451.

that significantly fewer tufts are found in sections ground with extreme care than are present in sections ground by the more ordinary methods, is at present under investigation by me, with early results pointing toward confirmation of Akamatsu's claims." In the discussion following the presentation of this paper, Dr. Chase, whose views oppose the occurrence of circulation between the dentin and

FIG. 1. THREE TUFTS SPRINGING FROM AMELO-DENTINAL JUNCTION

enamel, contended more pointedly that tufts are cracks. More recently White (7), working under Chase, published the results of their joint investigation of this subject. I call attention to the fact that the results of the research described in these two papers were ultimately the opposite of the outcome anticipated by Chase, for we read in White's conclusions that tufts are *not* artefacts. White's table of tuft incidence (p. 2375), which shows a remarkable uniformity of distribution, harmonizes with what I have said about tufts. But the

statement at the close of his paper, that "Beust's claims of uniform distribution and approximately constant numbers are not supported," bespeaks an inadequate acquaintance with my illustrations and descriptions (2, 3, 4); also a lack of knowledge of the structure of the tuft, and of the microscopic anatomy of the enamel. It is a very

FIG. 2. THE UNDER-SIDE OF ENAMEL SHELL OF BICUSPID SHOWING STAINED FISSURE-LIKE LINES (TUFT ROWS) MARKING POINTS OF ENTRANCE OF FIBRILS PASSING INTO STRUCTURE OF TUFTS

These lines extend from amelo-cemental junction to apex of crown. At *a*, supplemental line is seen. At *b*, adherent dentin covers continuation of rows.

simple procedure to count tufts, but to comprehend their true morphology and distribution is another matter. A tuft is a composite structure made up of numbers of characteristically curved fibrils arranged in a row. It is the aggregation of the individual fibrils that presents the image of the tuft, as an aggregation of trees exhibits the image of a grove. The number of tufts in a section depend, in a

measure, on its thickness. In the thinnest sections of enamel obtainable there are fewer and less characteristic tufts than in thicker sections, for the same reason that a thin slice out of a grove would not display the well known image of a grove, but only of trees here and there.

Viewed under a microscope, the tufts appear to arise from a common center (8). But if the anatomy and distribution of the tufts be

FIG. 3. HIGH-POWER VIEW OF PORTION OF UNDERSURFACE OF ENAMEL IN FIG. 2, SHOWING SEGMENTED STRUCTURE OF ROWS

Each fissure-like opening (hilum) is composed of linear series of tubes. Section at right angles to trend of rows would display, at position indicated by line, *a*, five tufts; at line, *b*, two tufts; at line, *c*, no tufts; at line, *d*, three tufts.

studied from the base of the enamel, in the region where it comes in contact with the dentin, as was done by me in 1924 (3), one sees that the tufts spring from the amelo-dentinal junction in parallel segmented rows, which run from close to the amelo-cemental junction over the apex of the crown (*fig. 2*; *study* the legends under *figs. 2* to 7). It is due to this segmented condition, also to deflections in the course

of the rows of fibrils and to differences in the spacing of the rows (all of which are visible in my published illustrations), that sections cut at right angles to them (i.e., cross-sections of *teeth*) exhibit gaps or differences in spacing. This is especially evident where the plane of section passes through the intersegmental spaces, or includes deviations from the common vertical course of the row, as may be seen in

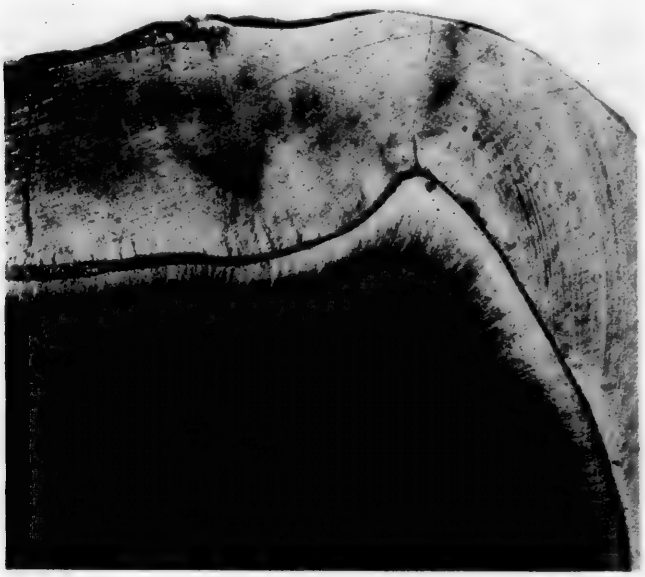

FIG. 4. LONGITUDINAL SECTION OF MOLAR WITH MORSAL AND CIRCUMFERENTIAL ENAMEL

Tufts are visible in morsal enamel only; those normally visible in circumferential enamel of cross sections of teeth being absent here, owing to angle at which section was cut.

fig. 3. This vertical, segmented arrangement of the parallel tuft-rows is not understood by Chase and White, although proof of it was submitted by me nearly ten years ago (2). I pointed out at that time that sections cut at right angles to these rows display characteristic tufts. Deviations, from this angle, display more or less distorted images, finally presenting in the longitudinal section, which follows the trend of the rows, only inconspicuous fragments, or no tufts at all (*figs. 4* and *5*). Exceptions to this rule may be found in the morsal enamel

of longitudinal sections, where the plane of the section bisects the tuft-rows at or near a right angle, as they pass over the crown (*fig. 6*). These facts explain why White failed to find tufts over line angles[2]

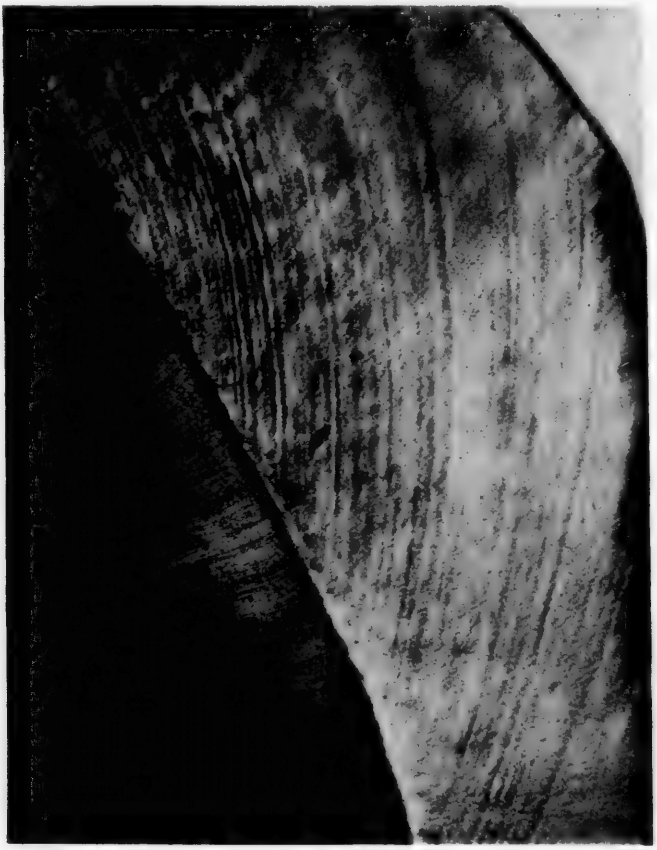

FIG. 5. HIGHER-POWER VIEW OF CIRCUMFERENTIAL ENAMEL IN FIG. 4, SHOWING ABSENCE OF CHARACTERISTIC TUFTS

and various other areas mentioned, where a variation in the angle of section might have disclosed them.

[2] "Line angle," as used by White (7), indicates the line where two surfaces join. Where three surfaces join, they form a "point angle."

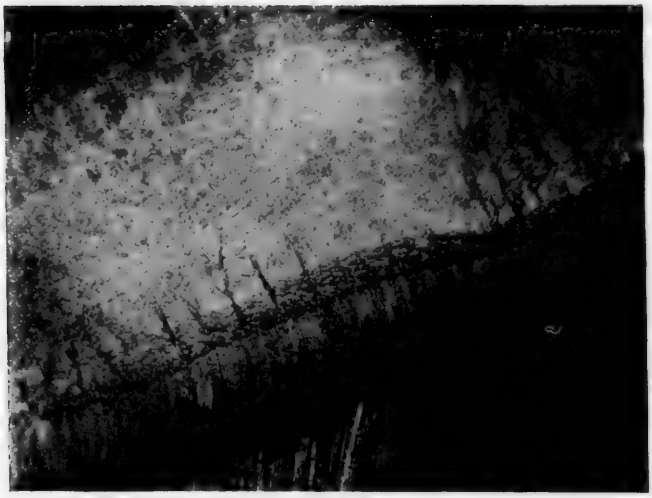

FIG. 6. HIGHER-POWER VIEW OF INNER REGION OF MORSAL ENAMEL IN FIG. 4, SHOWING BISECTED TUFT ROWS (TUFTS)

FIG. 7. TUFT ROWS CONVERGING ON MORSAL FISSURE OF LOWER MOLAR

It is regrettable that the arrangement and distribution of the tufts into rows has heretofore been overlooked by histologists. These rows profoundly modify the structure of the enamel in that they divide its inner regions into parallel compartments. Of this fact no mention has been made in the text-books.

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DENTAL CARIES EXPERIMENTALLY PRODUCED IN THE RAT[1]

S. N. BLACKBERG, PH.D., AND J. D. BERKE, D.D.S.

Department of Pharmacology, College of Physicians and Surgeons, and Department of Oral Histology and Embryology, School of Dental and Oral Surgery, Columbia University, New York City

Dental caries is the most widespread disease of civilized communities, affecting well over 90 percent of their population. Research hitherto has not been very fruitful either in determining the causative mechanism or in controlling the pathologic process, although many competent workers have studied the problem. It is generally agreed that in order to study the onset and progress of any disease, the lesion must be produced in experimental animals, so that all the variable factors can be controlled. This precept presents several essential problems: (*a*) the production of the lesion; (*b*) the characteristics of the experimentally produced pathology, and their resemblance to the human disease; (*c*) the consistent presence of the lesion in properly controlled groups of animals.

The rat has been used extensively in investigations on this problem. The low cost of the animal, the inexpensive feeding, the number that can be utilized in one experiment, and the adaptability of the animal to laboratory work, have accounted for its practically universal use in caries research. Although several workers (1) have described lesions in the teeth of rats, the macroscopic and histologic findings were not similar to human caries. Subsequent investigators (2), attempting to duplicate the reported lesions, have failed to produce an identical pathology. The diversity of causative mechanisms, and the various types of resulting lesions reported by previous investigators, have suggested the importance of conducting a series of carefully controlled researches.

In order to study the sequence of events in the development of

[1] Acknowledgment is hereby made of the financial assistance of the Commonwealth Fund in the pursuit of this study.

dental caries, we used large groups of rats and dogs, sacrificing and studying them at various stages. The earlier observations on the dentition of young animals on deficient diets were described in a preliminary report (3). Unquestionable pulpal reaction was noted in the animals used. In the rat, it was observed at 25 days of experimental feeding. The earliest notable variation was an extensive engorgement of the blood vessels. The odontoblasts became distorted and irregular in arrangement. As the process progressed there was vacuolation of these cells and gross cellular destruction. As this condition advanced there was concurrent change of the reticular cells of the pulp to fibroblasts, especially noticeable adjacent to the layer of Weil. This peripheral fibrosis seemed to be scar tissue replacing destroyed odontoblasts. The remaining pulpal tissue underwent various degenerative changes, the exact nature of which has not been determined. Vacuoles appeared in the pulpal tissue. The collagen content was increased. In more advanced conditions pulpal necrosis was seen. The sequelae to these conditions will be described in another paper in the succeeding issue of this *Journal*. The importance of these pulpal conditions lies in the finding that an extension of the period of experimental feeding produces dentin and enamel changes. This intense reaction within the tooth seems to cause a leaching out of the calcium salts of the calcified tissues, revealing the collagenous matrix of the dentin. This decalcification is readily evident radiographically (4). The final stage in the pathologic process is the destruction of the enamel.

The present communication is a study of the lesions in the molars of rats produced by disturbing the Ca-P metabolism. The animals were five weeks old when the experiment was initiated. Litter-mates were divided into three groups; basal rations, with a therapeutic content of vitamin D; basal rations, with an excessive amount of vitamin D; and basal rations, without vitamin D. The experiment was conducted for 150 days before the animals were sacrificed. The tissues were placed in 10 percent formalin immediately after death. The basal rations used were the modified Steenbock and Black Diet No. 2965.[2] The "excess" of viosterol was fed by pipette daily to the group

[2] Steenbock, H., and Black, A., *Journal of Biological Chemistry*, 1925, *64*, p. 263 (parts in 100): Yellow ground corn, 73; gluten, 20; Wesson Oil, 2; NaCl, 2; $CaCO_3$, 3; cod-liver oil (5 percent) added to the control diets.

that received it, the daily dose per rat at the initiation of the experiment having been one drop (0.05 cc.) of 250 D viosterol. This was gradually increased until, at the termination of the feeding time, the "excess" group received 0.8 cc. per day. The animals were kept in a constant state of hypercalcemia. The vitamin-D content of the normal-rat diet was equivalent to 5 percent of cod-liver oil. In order to prevent error due to the angle at which the tissues were cut, serial sections of the jaws were made. The ground specimens were embedded in Kollolith.

FIG. 1. Photomicrograph of first molar of rachitic rat. Note engorged blood vessels

The illustrations are from unretouched photomicrographs. The *control* group, receiving the basal rations with a therapeutic vitamin-D content, was free from dental caries. The pulps were vascular, but normal in structural detail. The *rachitic* group was fed basal rations alone, and presented a caries incidence of 33.3 percent. The lesions obtained involved the enamel, dentin, and pulp. Of the animals fed the basal rations with an *excess* of viosterol, all developed caries. As in previous observations (3), the incipient stage of the pathologic process was an engorgement of the blood vessels of the pulp. At this stage, teeth exhibiting this vascular change presented no demonstrable lesions in the dentin or enamel (*fig. 1*). With the continuation of this

congestion, a condensation of the reticular cells of the pulp was apparent, affecting the cells adjacent to the layer of Weil (*fig. 2*). Concurrent with this gradual metamorphosis was a definite odontoblastic destruction, beginning with distortion and vacuolation of the cells, and terminating, in extreme cases, in necrosis (*fig. 3*). In

FIG. 2. Photomicrograph showing hemorrhage in pulp, *A*. Note condensation of cells adjacent to layer of Weil, *B*.

instances where the pulps had not become necrotic, the odontoblasts were replaced by a layer of fibrous tissue. In the more advanced stages, the pulps underwent various degenerative changes. Vacuoles appeared throughout the tissue. The cells lost their definition, and the pulp assumed the appearance of a gelatinous mass (*fig. 3*). The pathology described above was localized to a portion of the pulp or involved the entire tissue. An unusual specimen is seen in *fig. 4*. The

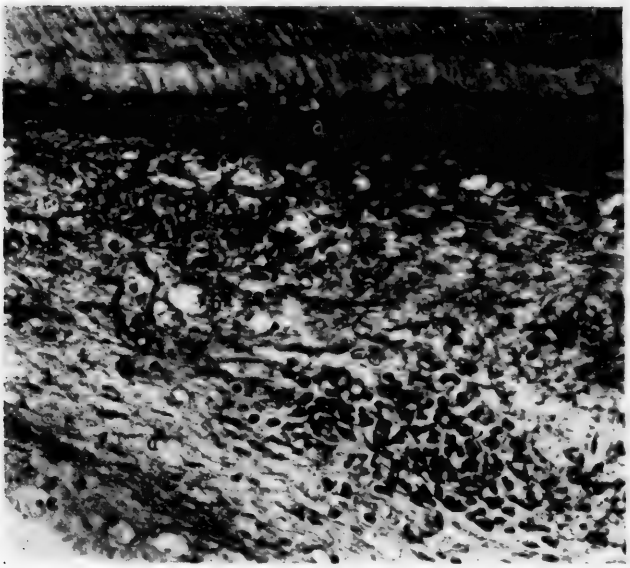

FIG. 3. Photomicrograph of pulp of rachitic rat. *a*, Fibrosed tissue. *b*, Degenerating reticular cells. *c*, Hemorrhages. *d*, Necrotic cells. *e*, Vacuolation of odontoblasts.

FIG. 4. Photomicrograph of first molar; excess-viosterol group. *a*, Mesial half of pulp, showing engorged blood vessels. *b*, Necrotic tissue. *c*, Carious process.

FIG. 5. Photomicrograph of molar of rat showing caries. *a*, Enamel. *b*, Dentin. *c*, Opaque dentin. *d*, Carious process. *e*, Pulp.

FIG. 6. Photomicrograph of rat molar showing caries. *a*, Enamel. *b*, Dentin. *c*, Carious dentin. *d*, Vegetable matter. *e*, Pulp.

mesial half of the tissue was vital, but greatly engorged. The distal portion was necrotic. The distal part of the occlusal surface was carious. The photograph indicates the greatest depth of the cavity seen in the serial sections.

In most instances, the characteristics of the carious lesions were clinically and histologically identical with the occlusal variety of

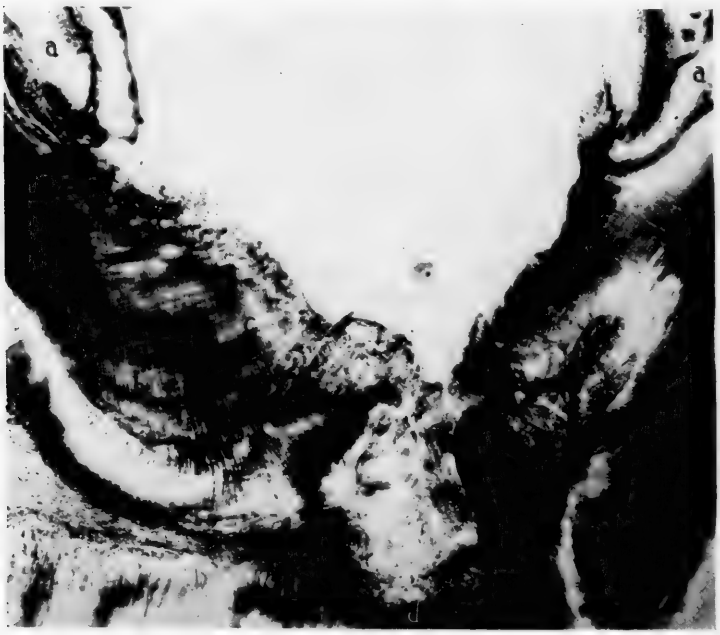

FIG. 7. Photomicrograph of rat molar showing extensive caries. *a*, Enamel. *b*, Dentin. C, Carious dentin. *d*, Pulp.

human caries. They occurred in the crevices of the occlusal surfaces of the molars, destroying the enamel and mushrooming out at the dento-enamel junction (*fig. 5*). With the progress of the destructive process, the adjacent enamel was undermined, and the penetration increased, producing the condition seen in *fig. 6*. The latter section is beyond the plane of the orifice of the cavity, and shows a vegetable inclusion from the diet. The pulp of the specimen seen in *fig. 7* had

been reached by the carious process. The cavities extend from the mesial to the distal cusps. The destruction had been so extensive (*fig. 8*) that the distal root was severed from the mesial half of the tooth. The pulp phenomena described above were visible in all carious specimens.

In the development of caries we have invariably noted an extreme engorgement in the blood vessels of the pulp. All other pathological

FIG. 8. Rat jaw showing extensive caries in first molar

changes follow this circulatory disturbance. There has been much speculation as to the significance of this sequence of events. That the circulatory disturbance can play an important rôle in decalcifying the hard tissues has been suggested in studies on rickets and osteoporosis. Our observations on the microscopic pathology, and the radiographic studies of the affected dental tissues, suggest a similarity of mechanism. This would indicate that a circulatory disturbance in the pulp, from any cause, might lead to a decalcification of the dentin and enamel. We are cognizant of the fact, however, that the

disturbance in the Ca-P metabolism which in our animals caused the pulpal changes might also be the agent directly responsible for the decalcifying process. External factors, such as mechanical, chemical, bacterial or physical processes in the mouth may influence the type or severity of the lesion, after decalcification has taken place. Experiments now in progress may clarify the relationship of these observed phenomena.

We express our gratitude to Dr. C. C. Lieb for his helpful guidance, to Dr. C. F. Bödecker for his valuable assistance in the interpretation of our findings, to Dr. E. Applebaum for the photomicrographs included, and to Miss N. Hughes for technical assistance.

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INCIPIENT DENTAL CARIES[1]

EDMUND APPLEBAUM, D.D.S.

Laboratory of Dental Histology and Embryology, School of Dental and Oral Surgery, Columbia University, New York City

A wealth of observation recorded in the literature attests the importance of the saliva in dental caries. The most obvious example, cited by Prinz (1), is the fact that, if the flow of saliva is impaired or completely checked, all the teeth will be destroyed by caries unless some other means for the removal of food debris is established. Thus in xerostomia, i.e., inhibition of secretion of saliva, the teeth begin to crumble away with the onset of dry mouth. Head (2), one of the earliest workers, reported experiments that "seem to show that partially softened enamel may have the power of becoming hard again. This rehardening is difficult to explain. . . . . The hardening may be a sort of recrystallization within the enamel substance; it may reunite with something taken from the saliva. I freely admit that I do not understand how it can happen, but I firmly believe that it does happen." Bunting (3) exposed unerupted teeth in a test tube for thirty days to the "high calcium salivas" of students immune to dental caries. One half of each tooth was saved for a control. Both halves were then stained in 10 percent silver nitrate solution and sectioned. Bunting states: "In each of these cases there was plainly seen in the original sections a marked difference in the penetration of the silver nitrate in the two halves, it being considerably less in the halves which had been carried in the high calcium salivas." Similar claims have been made by other workers, among whom is Andresen (4). In a recent monograph entitled, "The physiological and artificial mineralization of the enamel," he describes a method to judge the degree of mineralization and demineralization of the enamel by the use of polarized light. The polarizing microscope has not been widely used

[1] Acknowledgment is hereby made of the financial assistance of the Commonwealth Fund of New York in the pursuit of this study.

by dental histologists, yet it is a valuable aid in the study of the hard tissues of the tooth.

Thousands of ground sections of teeth have been studied with the compound microscope using ordinary light. The appearance is familiar to all histologists. Prof. Bödecker suggested a study of ground sections by the methods of the crystallographer. Through the courtesy of Prof. Kerr, of the Department of Mineralogy at Columbia University, the writer studied ground sections of teeth with the petrographic microscope. This instrument differs from an ordinary microscope in the addition of a rotating stage and of two Nicol prisms, one above and the other below the stage. Two Nicols are necessary, for the effects due to polarized light cannot be distinguished readily except by another Nicol. The lower Nicol is called the polarizer; the upper, the analyzer. The lower is set so that its vibration plane is at right angles to that of the upper Nicol. Then the field should be dark and the Nicols are said to be "crossed." If thin plates of singly refracting crystals are examined between crossed Nicols there is no result, for the original field remains dark. But if thin plates of doubly refracting crystals are examined between crossed Nicols, there result the beautiful color effects known as interference colors. These interesting optical phenomena are all due to the nature of light, and to the effect of the internal structure of certain crystals on light passing through them. With this apparatus, enamel, dentin, cementum, and bone, exhibit beautiful interference colors. Further, under convergent polarized light, enamel produces a biaxial interference figure characteristic of that class of crystals. Extinction is another optical phenomenon that can best be studied with a petrographic microscope fitted with a revolving stage. If a section of a doubly refracting crystal is revolved between crossed Nicols, there is darkness four times in a complete revolution. This is called extinction, and is simply due to the fact that for these particular positions the crystal has no effect upon the dark field produced by the crossed Nicols. It is rather significant that enamel possesses an extinction direction parallel to the course of the enamel rods. For in crystals, cleavage planes, which are determined by the regular internal arrangement of the atoms, are usually extinction directions. By trial it may be found that the particular interference-color given by any ground

section depends upon three factors: (*a*) double refraction, which is a constant for the crystal; (*b*) orientation or direction in which the crystal is cut; (*c*) thickness of the section. The enamel in cross section shows Newton's color-scale, first and second order, from the dento-enamel junction to the surface of the enamel. Thus dentin is white and the enamel exhibits yellow, red, blue, green, and yellow on concentric rings. The bands of Schreger, seen by reflected light in the enamel, are also apparent in polarized light. By means of the Becke test, used extensively by mineralogists, one can see that enamel has a high index of refraction—higher than dentin or the balsam in which it is embedded. This explains its brilliant appearance—exceeded, however, by the adamantine luster of the diamond.

That adult human enamel is largely made up of crystalline material seems to be the logical conclusion to draw from these studies. To test this belief, we powdered some enamel crowns in an agate mortar. All dentin was first removed with a bur. Then Prof. Kerr made an x-ray analysis of the enamel powder, and obtained a diffraction pattern on a strip of photographic film. His conclusion, from the diffraction pattern, was that beyond doubt it is a crystalline substance —50 percent or more. Another interesting point brought out by the x-ray analysis was the fact that a sample of fossil enamel, 900 years old, had exactly the same diffraction pattern as recent enamel. It became evident from these two methods of study that enamel is made up, in part at least, of crystalline material. The fact that enamel is doubly refracting, and that the individual crystals are not distinguishable even with a 4 mm. objective, indicates that adult human enamel is very finely divided and that the crystals approach the size of colloidal particles. This gives rise to the concept that the enamel is a dispersed system with crystal particles as one phase, and the organic matrix, first described by Bödecker, as the other.

The colloidal state or condition of enamel thus demonstrated may account for the intermittent character of incipient caries. That is to say, in this colloidal system there may be a demineralization of enamel by acid-forming bacteria followed by a remineralization of the same spot by the salts from the saliva. In incipient caries of the enamel an actual cavity has not yet been formed. Incipient caries is best studied on the enamel of the smooth surfaces of the teeth. Penetra-

FIG. 1. INCIPIENT CARIES OF ENAMEL UNDER POLARIZED LIGHT ×200

The color effects are not shown in the illustration. The band of demarcation between the carious enamel (above) and normal enamel (below) is shown by the broad black band.

FIG. 2. X-RAY OF GROUND SECTION OF TOOTH WITH INCIPIENT CARIES ON PROXIMAL SURFACE ×6
The carious spot, *C*, is seen to be radiolucent

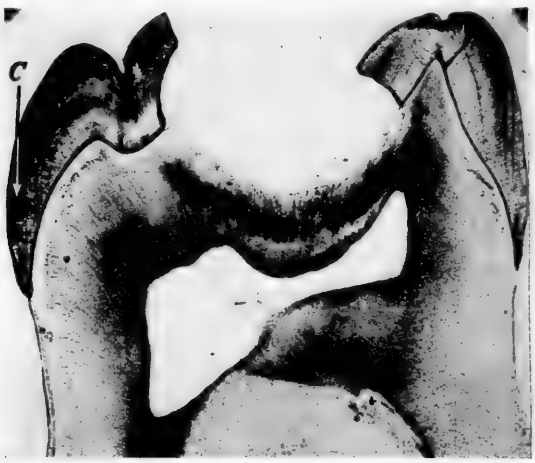

FIG. 3. PHOTOMICROGRAPH OF GROUND SECTION SHOWN IN X-RAY IN FIG. 2
The carious spot is seen at *C*

tion by acid follows the direction of the enamel rods; its advance is cone-like, with the apex toward the dentin and the base toward the surface of the tooth. Nevertheless the enamel is still so hard that the defect cannot be detected clinically with an instrument. It is also a well-known fact that different layers in the affected enamel in a ground section may be seen with ordinary transmitted light.

The different layers or zones in the enamel, seen in incipient caries, probably represent chemical changes due to diffusion of acid into the tissue. The organic matrix has also been affected, although some histologists would consider this to be merely the interprismatic cement substance. The changes seen in incipient caries are still more interesting when studied by polarized light. Different layers or zones are more definitely shown by various color effects familiar to students of minerals. The most striking zone is the band of demarcation, separating the outer cone of demineralized or carious enamel from the unaffected enamel (*fig. 1*). The carious or affected cone is of the same sign as the unaffected enamel; that is to say, they are both negative when tested by a mica plate inserted between the crossed Nicols. But the band of demarcation is of positive sign when so tested. This phenomenon has been observed by Andresen and confirmed in the writer's studies of the process with Prof. Kerr and Dr. Failey. The optical phenomenon therefore seems to justify Andresen's conception of remineralization of the enamel. In his paper he writes:

> "As these acids have diffused from the surface through the cuticula, the lamellae and the organic reticulum and on their way, have had the effect of making the enamel positive double refracting and as this part has again become negative, we have thereby a proof of the salivatoric remineralization. How would these changes in the double refraction be able to take place if the diffusion of the acid had not been followed by a diffusion and absorption of calcium salts?"

Andresen advocates the use of a powder to harden the enamel from the exterior, with this formula (amounts in grams): tartaric acid, 448; gelatine (10 per cent solution) 50—M. et. ad. Precipitated phosphate of calcium, precipitated carbonate of calcium, basic carbonate of magnesium, ana, 160—M., sic. et ad. Bicarbonate of sodium, 560, chloride of sodium, 160, corrigents of taste, ad libitum—M. d. s.

Effervescing, mineralizing, anticarieticum. The patient is instructed to apply, after thoroughly cleaning the teeth at night, a small quantity to those areas of the teeth to be treated, and to retain as much of this powder in the mouth during the night as possible. Bödecker (5) states:

"I have been using this method for about four years and have had excellent results in a number of cases. My application of this method has been more in the attempt to harden soft, caries-ravaged, sensitive teeth, and in this connection I have had marked success. I am convinced, however, that the treatment of white spots from the exterior is a mere make-shift. I believe that the natural process of furnishing the enamel with sufficient mineral matter is from the interior, i.e., by way of the pulp."

A chance observation which further contradicts the conception of remineralization from the exterior, i.e., by the saliva, is the x-ray picture (*fig. 2*) of a thin ground section of enamel showing incipient caries (*fig. 3*). Although it showed the characteristic polarizing phenomena, the little cone of affected enamel was more radiolucent than the normal, i.e., unaffected, enamel. This indicates a decrease in density of the caries cone in spite of "remineralization." How to reconcile the x-ray picture with the polarizing phenomena is indeed difficult. Without overlooking the importance of saliva in incipient caries of enamel, the appearance of different zones may be interpreted from the point of view of resistance to acid by enamel itself.

That the destruction of human enamel is not as simple as the dissolution of a lump of marble in a beaker of acid has been shown by the work of Bödecker (6). By the celloidin decalcifying method, specimens were obtained showing that enamel is "destroyed by two agencies: (a) by acid, in all probability lactic acid, formed by B. acidophilus, and (b) by the proteolytic action of bacteria (streptococci or diplococci?) upon a portion of the protein content. A novel and interesting factor in these specimens is the occurrence of a zone between normal and diseased enamel that takes the Heidenhain ferric hematoxylin stain most intensely. This area, which is noted as an intensely dark zone, separates the normal from the infected tissue, and appears to be a form of degeneration of the organic matrix preparatory to its destruction by the bacteria. This zone has never been seen before (as adult

enamel cannot be decalcified by ordinary methods), and the authors have named it the "zone of carious degeneration."

Another important finding has been recorded by Mummery (7). He studied incipient caries in deciduous molars. The teeth were preserved in sublimate and stained with borax carmine in bulk. They were prepared by the Weil method. In the first twenty cases prepared, showing the spots of decay confined to the enamel, twenty-four distinct zones of translucency were found. Carmine stains the carious portion very deeply, and its margin is sharply defined by the clear zone beneath it. In two of these twenty cases the decay, although it had not reached the dentin, advanced in narrow-pointed processes, apparently indicating very rapid penetration, and no zones were visible. But they were very distinct where the caries had advanced slowly, as indicated by the rounded lower margin of the diseased portion. Quoting from Mummery (7):

"We have now to consider what is the nature of this zone. It is evidently not an area of decalcification for the stain does not enter it but ends abruptly at its margin. Also in specimens where the zone involves the more open structure of the striae of Retzius, these appear to be partially obliterated and have almost or quite disappeared in the clear area. These facts point to the probability of the translucent zone being an area of denser calcification than the rest of the enamel and closely correspond to the similar evidences of the nature of the translucent zone in the dentin.

"There has been from time to time some controversy about the meaning of the translucent zones in the dentin but it is now, we think, very generally accepted that they are the sole indication of vital reaction on the part of the pulp in caries—they do not occur in dead teeth. These zones in the dentin serve at all events as a temporary barrier to the advance of the microörganisms of decay; they not only occur in caries but also as a response to irritation from attrition and they are seen in arrested caries where they seem to form a more resistant surface to wear. These areas in the dentin stain very imperfectly and in many cases not at all. When the capillary attractions method is used with alcoholic fuchsin (a very penetrating stain) they usually remain quite uncolored and there is every reason to believe that there is a deposition of calcific material in and around the tubules in these areas.

"If such a deposition of lime salts can take place in the dentin it seems highly probable that a similar deposition can occur in the enamel as the

dentinal tubes are in many places, more particularly at the bundles or whorls, so conspicuous at the dentino-enamel junction, continuous with tubular spaces in the enamel.

"Whatever this alteration in the enamel may be there appears to be a distinct change of structure which is *not due to decalcification* and is intimately associated with the decayed area, indicating that enamel cannot be a dead inert substance but is capable of reaction to stimuli and therefore, like other tissues of the body, endowed with vitality."

The conclusion that may be drawn from this study is that while the lesion of incipient caries of the enamel is essentially a decalcification within an organic matrix, yet a zone which is very often present, and called by various names, is the key to the situation. This band of demarcation between decayed and normal enamel seems to be, from work done by Charles F. Bödecker and the present writer, similar to the well-known translucent zone in the dentin. The formation of such a barrier in enamel may indeed decide the arrest of the carious process.

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REPORT OF A TABULATION OF DENTAL STATE BOARD EXAMINATIONS FOR 1931[1]

H. E. FRIESELL, *Chairman of the Tabulation Committee*[2]

University of Pittsburgh, Pittsburgh, Pa.

The Tabulation Committee has audited the State Board reports for 1931, and presents herewith its report. Reports for forty-eight (48) states were received and tabulated.

The total number of dental graduates in the United States for the year 1931 was 1842, an increase of 96 over 1930. Of this number, 1718 were examined by the State Boards reporting: number passed, 1595; number failed, 123; percentage of failures, 7.2—an increase of 1.4 percent over that of the previous year. The total number of graduates reported for the twenty-two (22) years of tabulation—1910 to 1931, inclusive—is 53,583: total examined, 46,622; passed, 40,682; failed, 5,940; percentage of failures, 12.74. It is a pleasure to report that of thirty-nine (39) schools, ten (10) had no failures in 1931, and eight (8) had less than 7.2 percent (the general total average for the year). The Committee notes a continued interest on the part of the schools in keeping informed about the success or failures of their students.

Appended hereto is a detailed statement showing the results of the tabulation of State Board reports for graduates of the year 1931 (*table 1*), and also the cumulative tabulation for the years 1910 to 1931, inclusive (*tables 2* and *3*). Some State Boards still show a much larger percentage of failure than others. Attention is directed to the following Boards whose reports for 1931 cover more than twenty (20) applicants—graduates of 1931:

New Jersey	72.0	percent	failed of	25	examined
Minnesota	19.4	"	" "	62	"

[1] Presented at the Ninth Annual Meeting of the American Association of Dental Schools, Columbus, O., March 21–23, 1932.

[2] The Committee, Drs. H. E. Friesell and George L. Powers, serve the Dental Educational Council of America, the American Association of Dental Schools, and the National Association of Dental Examiners.—[*Ed.*]

TABLE 1

Results of tabulation of State Board reports for graduates for the year 1931

STATE	COLLEGE	TOTAL NO. OF GRADUATES	TOTAL NO. EXAMINED	EXAMINED: PASSED	EXAMINED: FAILED	PERCENTAGE OF FAILURES	NO. OF STATES IN WHICH GRADS. WERE EXAMINED
Cal.	U. of Calif. College of Dent.	47	43	37	6	14.0	1
	Col. of P. & S. School of Dent.	36	36	33	3	8.3	1
	U. of Southern Calif. Col. of Dent.	94	94	84	10	10.6	3
Colo.	U. of Denver, School of Dent.	12	12	11	1	8.3	2
D. C.	Howard University, Col. of Dent.	12	3	2	1	33.3	3
	Georgetown University, School of Dent.	21	19	9	10	52.6	6
Ga.	Atlanta-Southern Dental College	54	53	50	3	5.7	10
Ill.	Chicago College of Dental Surgery	108	101	91	10	9.9	6
	Northwestern University Dent. School	75	64	59	5	7.8	7
	University of Illinois, Col. of Dent.	46	41	37	4	9.7	5
Ind.	Indiana University, School of Dent.	31	31	31	—	—	6
Iowa	State University of Iowa, Col. of Dent.	32	32	32	—	—	1
Ky.	University of Louisville, School of Dent.	37	36	31	5	13.9	10
La.	Loyola University of La., School of Dent.	22	21	18	3	14.3	5
Md.	Baltimore College of Dent. Surgery	54	47	42	5	10.6	7
Mass.	Tufts College Dental School	43	43	38	5	11.6	6
	Harvard University Dental School	19	15	13	2	13.3	4
Mich.	University of Michigan, School of Dent.	76	74	71	3	4.0	1
Minn.	University of Minnesota, College of Dent.	79	72	61	11	15.3	5
Mo.	Kansas City-Western Dental College	45	36	36	—	—	4
	Washington University, School of Dent.	40	39	39	—	—	4
	St. Louis University, School of Dent.	63	59	54	5	8.5	14
Nebr.	University of Nebraska, Col. of Dent.	28	28	28	—	—	5
	Creighton University, Col. of Dent.	21	21	21	—	—	5
N. Y.	New York University, College of Dent.	76	72	70	2	2.8	1
	University of Buffalo, School of Dent.	23	22	20	2	9.1	1
	Columbia University, School of D. & O. S.[1]	80	78	73	5	6.4	2
Ohio	Western Reserve University, Sch. of Dent.	21	21	21	—	—	1
	Ohio State University, Col. of Dent.	69	64	64	—	—	2
Oregon	No. Pacific Col. of Oregon, S. of D.	43	26	23	3	11.5	4
Penna.	Temple University Dental School	107	107	105	2	1.9	4
	University of Pittsburgh, School of Dent.	115	114	113	1	0.9	1
	University of Penna., School of Dent.	50	44	36	8	18.2	5
Tenn.	University of Tennessee, Col. of Dent.	30	30	29	1	3.3	2
	Meharry Med. Col. Dept. of Dent.	16	9	8	1	11.1	6
Texas	Baylor University, College of Dent.	12	12	12	—	—	1
	Texas Dental College	24	21	18	3	14.3	2
Va.	Medical College of Va., School of Dent.	21	20	20	—	—	3
Wisc.	Marquette University Dental School	60	58	55	3	5.2	3
Total		1,842	1,718	1,595	123	7.2	

Summary:

Number of State Board reports received	48
Number of State Board reports tabulated	48
Total number of applicants examined and passed	1,595
Total number of applicants examined and failed	123
Percentage of failures	7.2

[1] The Dean of this School refused to coöperate in verifying the figures.

TABLE 2

Cumulative results of tabulation of State Board reports for graduates of existing schools for the years 1910 to 1931, inclusive

STATE	COLLEGE	TOTAL NO. OF GRADUATES	TOTAL NO. EXAMINED	EXAMINED: PASSED	EXAMINED: FAILED	PERCENTAGE OF FAILURES	NO. OF STATES IN WHICH GRADS. WERE EXAMINED
Cal.	University of Calif. Col. of Dent.	1,036	800	744	56	7.0	4
	Col. of P. & S. School of Dent.	912	644	556	88	13.7	4
	University of So. Calif. Col. of Dent.	1,558	1,229	1,110	119	9.7	10
Col.	University of Denver, School of Dent.	732	686	613	73	10.6	20
D. C.	Howard University Dental School	637	286	193	93	32.5	31
	Georgetown University, Dental Dept.	537	434	328	106	24.4	27
Ga.	Atlanta-Southern Dental College	1,066	1,041	1,004	37	3.6	18
Ill.	Chicago College of Dental Surgery	2,773	2,400	2,128	272	11.3	36
	Northwestern Univ. Dental School	2,413	1,982	1,768	214	10.8	40
	University of Illinois, Col. of Dent.	762	676	620	56	8.3	24
Ind.	Indiana University, School of Dent.	1,347	1,295	1,182	113	8.7	25
Iowa	State University of Iowa, Col. of Dent.	1,186	1,142	1,095	47	4.1	17
Ky.	Univ. of Louisville, School of Dent.	818	751	677	74	9.9	32
La.	Loyola Univ. of La., School of Dent.	237	212	199	13	6.1	15
Md.	Baltimore Coll. of D. S., Univ. of Md.	1,420	1,217	958	259	21.3	27
Mass.	Tufts College Dental School	1,810	1,623	1,449	174	10.7	14
	Harvard University Dental School	926	740	657	83	11.1	18
Mich.	University of Michigan, Col. of Dent.	1,766	1,630	1,590	40	2.5	20
Minn.	University of Minn. College of Dent.	1,696	1,516	1,416	100	6.5	17
Mo.	Kansas City-Western Dental College	844	743	718	25	3.4	19
	Washington Univ., School of Dent.	648	618	598	20	3.2	17
	St. Louis University, School of Dent.	1,282	1,190	1,122	68	5.7	32
Nebr.	Univ. of Nebraska, College of Dent.	439	404	383	21	5.2	14
	Creighton University, College of Dent.	618	592	546	46	7.8	17
N. Y.	New York Univ., College of Dent.	3,053	2,789	2,096	693	24.8	7
	University of Buffalo, School of Dent.	965	891	707	184	20.7	8
	Columbia Univ., School of D. & O. S.[1]	679	654	523	131	20.0	4
Ohio	Western Reserve U., School of Dent.	812	774	694	80	10.3	6
	Ohio State Univ., College of Dent.	877	841	795	46	5.5	13
Oregon	No. Pacific Col. of Oregon S. of D.	1,677	1,348	1,038	310	23.0	19
Penna.	Temple University, School of Dent.	1,446	1,259	1,089	170	13.5	22
	Univ. of Pittsburgh, School of Dent.	2,236	2,157	2,093	64	3.0	16
	University of Penna., School of Dent.	3,297	2,507	2,046	461	18.4	29
Tenn.	University of Tenn. College of Dent.	369	349	328	21	6.0	16
	Meharry Med. Coll., Dept. of Dent.	843	676	508	168	24.9	32
Texas	Baylor University, College of Dent.	546	412	372	40	9.7	10
	Texas Dental College	440	266	216	50	18.8	5
Va.	Med. Coll. of Virginia, Sch. of Dent.	363	346	326	20	5.8	9
Wisc.	Marquette University Dental School	1,638	1,504	1,402	102	6.7	24
Total		46,704	40,624	35,887	4,737	11.7	
Totals for Schools discontinued (Table 3)		6,879	5,998	4,795	1,203	20.0	

Summary for active schools: Total number of applicants examined and passed. 35,887
Total number of applicants examined and failed... 4,737
Percentage of failures..... 11.7

[1] The Dean of this School refused to coöperate in verifying the figures for the examinations in 1931.

Kentucky	16.7	percent	failed	of	24	examined
Texas	13.6	"	"	"	44	"
Michigan	13.2	"	"	"	91	"
California	11.1	"	"	"	171	"
New York	9.1	"	"	"	208	"

In thirty (30) states there were no failures.

The figures shown in the tabulations represent the results of the first examination taken by graduates in the year of graduation, including the examinations subsequent to the regular summer examination up to December 31, 1931. A few errors on the part of State Board secretaries came to light when the colleges were asked to confirm the correctness of the failures listed against them. It was found that some applicants had been incorrectly reported as failed, according to the college statement. Any adjustment will be made in the cumulative record for next year, when the secretaries of the State Boards have replied to our inquiries. Attention is called to an error in the tabulation proceedings of the Eighth Annual Meeting of the American Association of Dental Schools. On page 92, tabulation for 1930; College of Physicians and Surgeons, California: the percentage of failures should read 15.4 instead of 6.7.

Your committee is gratified with the continued evidence of coöperation on the part of the schools and the State Board secretaries, and wishes to record appreciation of the assistance which enables it to procure the information necessary to make the tabulation valuable as a record.

Because of a limitation of funds and facilities available to the Tabulation Committee, the reports in the past have presented only the results of the tabulations as they pertained to the schools. At the meeting last year, it was considered advisable to have the Tabulation Committee also report the figures as they applied to the various State Boards. Therefore, there is appended to this report a detailed statement showing the number of graduates examined, and the number of failures before each State Board, for the cumulative period 1910 to 1931, inclusive (*table 4*). This table shows the figures covering the examinations by each State Board for the total period of twenty-two (22) years. These figures, of course, refer only to the result of the applicant's first appearance before the State Board during the calendar year in

which he graduated. Likewise they cover only the first Board the applicant is examined by during that calendar year. For instance—if fifteen men were examined by the Alabama Board in 1931 and a week or two later ten of these fifteen individuals and five others took the Cali-

TABLE 3

Cumulative results of tabulation of State Board reports for graduates of dental schools that were discontinued since 1910 and before 1931

STATE	COLLEGE	TOTAL NO. OF GRADUATES	TOTAL NO. EXAMINED	EXAMINED: PASSED	EXAMINED: FAILED	PERCENTAGE OF FAILURES	NO. OF STATES IN WHICH GRADS. WERE EXAMINED
Ala.	Birmingham Dental College	54	50	38	12	24.0	1
D. C.	Geo. Washington Univ. Dept. of Dent.	185	137	108	29	21.2	13
Ga.	Southern Dental College	362	337	303	34	10.0	10
	Atlanta Dental College	472	441	367	74	16.8	15
	So. Eastern Dental Univ.	2	2	—	2	100.0	2
Iowa	Drake Univ. Col. of Dent.	32	29	29	—	—	1
La.	Louisiana State Col. of Dent. Surg.	6	—	—	—	—	—
	Tulane Univ. of La. School of Dent.	405	333	314	19	5.7	14
Md.	Balto. Col. of Dental Surgery	775	564	392	172	30.5	23
	Balto. Medical Col. Dental Dept.	83	64	43	21	32.8	12
Mo.	Kansas City Dental College	514	470	434	36	7.7	14
	Western Dental College	456	413	366	47	11.4	18
	Barnes School of Dent.	82	70	55	15	21.4	5
N. J.	College of Jersey City Dental Dept.	57	53	7	46	86.8	1
N. Y.	College of D. & O. Surg. N. Y.	1,457	1,367	976	391	28.6	6
Ohio	Ohio College of Dental Surgery	655	572	445	127	22.2	23
	Cincinnati Col. of Dental Surgery	227	195	138	57	29.2	11
Penna.	Med. Chi. College Dental Dept.	348	248	176	72	29.0	9
Tenn.	Vanderbilt Univ. School of Dent.	613	574	544	30	5.2	22
	Univ. of Memphis, Denta. Dept.	5	4	4	—	—	3
	Univ. of W. Tenn. Col. Dent. Surg.	40	28	18	10	35.7	5
Va.	Univ. Col. Medicine, Dental Dept.	32	30	23	7	23.3	2
Wisc.	Wisc. Col. Physicians & Surgeons	17	17	15	2	11.8	1
Total		6,879	5,998	4,795	1,203	20.0	

fornia Board examinations, only the "five others" would be tabulated in the California report, as these figures do not include second or third examinations. If time permits, the Committee hopes to present next year a tabulation of the various State Board results in a manner that will give additional information.

TABLE 4

State Board records: 1910–1931, inclusive; cumulative record of the examinations in the individual states

STATE	TOTAL NUMBER EXAMINED	TOTAL NUMBER OF FAILURES	PERCENTAGE OF FAILURES
Alabama	250	36	14.4
Arizona	13	4	30.8
Arkansas	152	11	7.2
California	2,677	280	10.4
Colorado	649	60	9.2
Connecticut	353	165	46.7
Delaware	46	10	21.7
District of Columbia	366	63	17.2
Florida	135	10	7.4
Georgia	1,439	111	7.7
Idaho	67	16	23.9
Illinois	4,339	334	7.7
Indiana	1,392	135	9.7
Iowa	1,371	153	11.2
Kansas	624	8	1.3
Kentucky	658	52	7.9
Louisiana	533	11	2.1
Maine	144	5	3.5
Maryland	1,103	143	13.0
Massachusetts	2,239	267	11.9
Michigan	1,742	72	4.1
Minnesota	1,691	196	11.6
Mississippi	140	18	13.0
Missouri	2,321	81	3.5
Montana	103	18	17.5
Nebraska	856	51	6.0
Nevada	4	1	25.0
New Hampshire	82	18	22.0
New Jersey	824	581	70.5
New Mexico	20	2	10.0
New York	6,357	1,535	24.1
North Carolina	312	65	20.8
North Dakota	93	17	18.3
Ohio	2,351	312	13.3
Oklahoma	208	15	7.2
Oregon	855	180	21.1
Pennsylvania	4,967	298	6.0
Rhode Island	143	26	18.2
South Carolina	173	27	15.6
South Dakota	107	33	31.1
Tennessee	744	25	3.3
Texas	950	136	14.3
Utah	202	39	19.3
Vermont	42	8	19.0
Virginia	440	59	13.4
Washington	502	136	27.0
West Virginia	297	45	15.2
Wisconsin	1,493	67	4.5
Wyoming	33	2	6.1
Porto Rico	19	4	21.0
Totals	46,621	5,941	12.74

The present statement shows that for the twenty-two (22) years tabulated, eighteen (18) States have had less than ten percent (10%) of failures; nineteen (19) States have had between ten (10) and twenty (20) percent of failures; nine (9) States have had between twenty (20) and thirty (30) percent of failures; one (1) State—Arizona—examined thirteen (13) applicants who had not previously been examined by other Boards; of this number (4) were failed, making a percentage of failures of 30.8. One State—South Dakota—examined one hundred seven (107) applicants under similar conditions and failed thirty-three (33), making percentage of failures of 31.1. One State—Connecticut—examined three hundred fifty-three (353) applicants under these conditions, failing one hundred sixty-five (165) and showing a percentage of failures of 46.7. One State—New Jersey—examined eight hundred twenty-four (824) applicants under these conditions, failing five hundred eighty-one (581) of this number, with a percentage of failures of 70.5.

EFFECT OF DIET ON THE ACID-NEUTRALIZING POWER OF SALIVA

J. C. FORBES, M.A., PH.D., AND W. B. GURLEY, D.D.S.

Departments of Biochemistry and Operative Dentistry, Medical College of Virginia, Richmond, Va.

These experiments were conducted to determine whether the acid-neutralizing power of saliva could be influenced by diet, and if possible to ascertain the factors involved. For this purpose a number of subjects were placed on controlled diets, and the salivary acid-neutralizing power determined from time to time. In the preliminary series of experiments (*table 1*), the subject, *J. C. F.*, went without breakfast on the days of salivary examination, and samples were collected at various periods during the morning. In all other cases, breakfast was taken not later than 8:30 a.m., and saliva samples were collected at 11:45 and 12:45, ±15 minutes. These values, especially those obtained at the later period, are undoubtedly close to the maximum value reached by this individual on that day. They were collected sufficiently long after breakfast for the influence of that meal on the salivary acid-neutralizing action, as reported in a subsequent publication (1), to have disappeared. The results obtained on different diets should therefore be strictly comparable. The methods of analysis will be described in the above-mentioned publication (1).

All subjects chosen for these experiments are in their twenties, with the exception of *J. C. F.*, who is in his late thirties. Subjects *J. C. F.* and *C. P.* (*tables 1* and *2*, respectively) are at present relatively immune to caries, but heretofore have had a number of cavities. Subject *R. C. M.* (*table 3*) has exceptionally good teeth, and has had no cavities in recent years. Subjects *R. J.* and *O. O.* (*tables 4* and *5*, respectively) are quite susceptible at the present time. Subject *T. G. F.* (*table 6*) has rampart decay. The diets indicated in *tables 2*, *3*, *4*, and *5* were more accurately controlled than those in *tables 1* and *6*. In all cases the individuals were kept on the specified diet throughout the dietary period, even though the saliva was not analyzed every day.

In the preliminary series (*table 1*) the duration of each dietary period was only about a week—not long enough for the maximum effect of the diet to be manifested. Several days on a regular well-balanced

TABLE 1

Effect of diet on the neutralizing action of saliva (J. C. F.; relatively immune)

EXP. NO.	HCl ADDED cc.	pH AT TIME OF COLLECTION 7:30	8:30	9:00	9:30	10:30	12:00	REMARKS
1	0	6.9	6.9	6.9	6.9	6.8	6.9	Diet of meat, bread and cereals; no breakfast
	1.0	6.0	6.2	6.1	6.4	6.4	6.3	
	0.5	5.1	5.2	5.2	5.4	5.6	5.5	
2	0	6.8	6.9	7.0	6.9	6.9	6.9	Same as above
	1.0	6.0	6.2	6.0	6.0	6.1	6.2	
	0.5	5.0	5.4?	5.0	5.1	5.4	5.5	
3	0	6.7	6.7	6.7	6.8	6.8		Same as above
	1.0	5.8	6.0	5.9	5.8	6.0		
	0.5	4.9	5.0	4.9	4.8	5.2		
4	0	6.7	6.9	6.8	6.8	6.8	6.8	Same as above
	1.0	5.8	6.3	6.1	5.7	5.9	5.8	
	0.5	5.1	5.7	5.4	4.9	5.2	5.1	
5	0	6.7	6.9	6.8	6.8	6.8		Diet of vegetables, fruit, and meat, with a little bread; no breakfast
	1.0	5.7	6.1	6.1	6.2	6.1		
	0.5	4.9	5.1	5.1	5.4	5.3		
6	0	6.7	6.8	7.0	6.8	6.9	6.7	Same as above
	1.0	5.8	6.1	6.2	6.1	6.2	5.8	
	0.5	4.7	5.1	5.2	5.2	5.2	5.0	
7	0	6.8	6.8	6.8	6.9	6.8	6.9	Same as above, excepting that two oranges were taken at 10 a.m.
	1.0	6.2	6.1	6.0	6.1	6.0	6.1	
	0.5	5.3	4.9	5.1	5.2	5.2	5.4	
8	0	6.6	6.9	6.9	6.9	6.9		Diet of rice, bread and ice cream; no breakfast
	1.0	5.4	5.9	5.9	5.9	5.9		
	0.5	4.7	4.8	4.8	4.8	5.0		
9	0	6.5	6.9	6.9	6.9	6.6		Same as above
	1.0	5.0	6.0	5.8	5.8	5.4		
	0.5	4.2	4.9	4.7	4.8	4.5		
10	0	6.5	6.7	6.7	6.6			Same as above
	1.0	5.3	5.6	5.3	5.6			
	0.5	4.7	4.6	4.5	4.7			

diet were allowed to intervene between the special dietary periods. Tea or coffee, containing a small amount of milk, was taken at each meal. Breakfast was omitted only on the days on which the saliva was examined. The special diets (*tables 2, 3, 4,* and *5*) were these:

TABLE 2

Effect of diet on the neutralizing action of saliva (C. P.; relatively immune)

DATE	HCl ADDED	TIME OF COLLECTION				REMARKS
		11:45		12:45		
	cc.	pH	P: mg. per 100 cc.	pH	P: mg. per 100 cc.	
Oct. 30	0	6.8				Regular diet
	1.0	6.2				
	0.5	5.8				
Nov. 3	0			7.0		Diet as above
	1.0			6.3		
	0.5			5.2?		
" 4	0	6.9		6.9		Diet as above
	1.0	6.2		6.3		
	0.5	5.7		5.5		
" 6	0			6.9	9.5	Diet as above
	1.0			6.2		
	0.5			5.8		
" 11	0	6.9	14.0	6.9	13.2	Diet 1, started Nov. 7
	1.0	5.6		5.8		
	0.5	5.1		5.2		
" 13	0	6.7	11.5	6.8	11.6	Diet as above
	1.0	5.5		5.7		
	0.5	5.0		5.2		
" 17	0	6.9	16.0	6.9	15.5	Diet as above; small amount of meat and milk added, Nov. 14
	1.0	5.8		5.7		
	0.5	5.1		5.2		
" 18	0	6.8		6.9	13.2	Diet as above; small amount of meat and milk added
	1.0	5.9		6.1		
	0.5	5.4		5.5		
" 20	0			6.9	15.7	Diet as above
	1.0			6.0		
	0.5			5.3		
" 24	0	5.8	14.4	6.4	15.4	Diet 3, started Nov. 21; severe cold developed late on Nov. 20—still active on 24th
	1.0	4.7		4.6		
	0.5	4.3		4.3		
" 25	0	6.5	14.7	6.7	13.9	Diet as above; cold considerably better
	1.0	5.5		4.6		
	0.5	4.6		4.6		
Dec. 4	0	6.7		6.5	16.8	Diet as above; cold about same as above
	1.0	5.9		5.7		
	0.5	4.7		5.0		
" 8	0	6.9	17.6	6.8	17.9	Diet as above; cold practically over
	1.0	6.0		6.1		
	0.5	5.0		5.2		
" 9	0	6.9	15.3	6.9	15.2	Diet as above
	1.0	5.7		5.9		
	0.5	5.0		5.4		
" 11	0	6.8	16.0	6.9	14.0	Diet as above; fresh cold beginning
	1.0	5.2		—		
	0.5	4.4		5.1		

TABLE 3

Effect of diet on the neutralizing action of saliva (R. C. M.; exceptionally good teeth; no active caries)

DATE	HCl ADDED	TIME OF COLLECTION				REMARKS
		11:45		12:45		
	cc.	pH	P: mg. per 100 cc.	pH	P: mg. per 100 cc.	
Oct. 30	0	7.0		6.7		Regular diet
	1.0	6.5		6.4		
	0.5	6.4		5.8		
Nov. 3	0	7.0		7.0		Same as above
	1.0	6.3		6.0		
	0.5	6.0		5.7		
" 6	0	7.1	13.1			Same as above
	1.0	6.7				
	0.5	6.2				
" 7	0	6.9		7.1	13.4	Diet 1, started Nov. 7
	1.0	6.3		6.4		
	0.5	6.1		6.1		
" 11	0	7.1	10.3	6.9	11.6	Same as above
	1.0	6.5		6.3		
	0.5	6.3		—		
" 13	0			6.9	11.6	Same as above
	1.0			6.2		
	0.5			5.7		
" 17	0	6.9	13.5	6.9	14.2	Same as above
	1.0	6.3		6.3		
	0.5	6.1		5.8		
" 18	0	7.0	12.9	7.0	15.2	Same as above
	1.0	6.3		6.3		
	0.5	5.8		5.7		
" 20	0	6.9	15.1	6.9	15.3	Same as above
	1.0	6.1		6.3		
	0.5	5.3		5.3		
" 24	0	6.8	16.2	6.9	15.6	Diet 2, started Nov. 21; headache and slight fever, unknown origin, begun Nov. 23
	1.0	6.2		6.0		
	0.5	5.3		5.2		
" 25	0	6.9	14.6	7.1	14.3	Diet as above; fever continued
	1.0	6.3		6.3		
	0.5	5.9		5.7		
Nov. 25. Experiment discontinued until subject recovered						
Dec. 8	0	7.2	15.0	7.1	15.4	Diet as above; feeling quite well but weak
	1.0	6.6		6.4		
	0.5	6.3		5.7?		
" 9	0	7.1	14.0			Diet as above
	1.0	6.6				
	0.5	6.1				
" 11	0			7.2	13.6	Diet as above
	1.0			6.4		
	0.5			5.7		
" 15	0	7.0		7.0		Diet as above
	1.0	6.4		6.3		
	0.5	5.7		5.7		

TABLE 4

Effect of diet on the neutralizing action of saliva (R. J.; susceptible to caries)

DATE	HCl ADDED	TIME OF COLLECTION				REMARKS
		11:45		12:45		
	cc.	pH	P: mg. per 100 cc.	pH	P: mg. per 100 cc.	
Oct. 30	0			6.6		Regular diet
	1.0			5.2		
	0.5			4.5		
Nov. 3	0	6.8		6.9		Diet as above
	1.0	5.5		5.3		
	0.5	4.4		4.3		
" 4	0	6.7	9.5	6.7	10.6	Diet as above
	1.0	5.3		5.6		
	0.5	4.3		4.6		
" 6	0	6.5	8.5	6.6	8.6	Diet as above
	1.0	5.6		5.6		
	0.5	4.7		4.7		
" 10	0	6.6	12.7	6.8	10.0	Diet 2 started Nov. 7
	1.0	5.4		5.5		
	0.5	4.9		4.8		
" 13	0	6.6	10.5	6.8	11.1	Diet as above; urine alkaline Nov. 11–13; Nov. 14 diet 3 started
	1.0	5.4		5.7		
	0.5	4.9		5.1		
" 17	0	6.7	10.1	6.8	11.1	Diet 3 taken
	1.0	5.5		5.7		
	0.5	4.7		4.9		
" 18	0	6.7	9.4	6.7	10.2	Diet as above
	1.0	5.6		5.5		
	0.5	4.9		4.8		
" 24	0	6.8	9.8	6.8	10.0	Diet as above
	1.0	5.4		5.7		
	0.5	4.7		5.0		
" 25	0	6.7	11.0	6.9	10.4	Diet as above
	1.0	5.7		5.6		
	0.5	5.0		4.9		
Dec. 2	0	6.6		6.9		Diet as above
	1.0	5.5		6.0		
	0.5	4.7		5.3		
" 8	0	6.9	9.9	6.9	10.5	Diet as above
	1.0	5.9		6.0		
	0.5	4.9		5.3		
" 9	0	6.9	9.7	6.9	8.8	Diet as above
	1.0	6.0		6.1		
	0.5	5.1		5.0		
" 11	0	6.9	10.0	6.9	9.8	Diet as above; fairly severe cold
	1.0	6.0		6.0		
	0.5	5.1		5.2		
" 15	0	6.9	9.8	6.9	9.9	Diet as above
	1.0	5.7		5.8		
	0.5	4.8		5.1		
" 16	0	6.8		6.9		Diet as above
	1.0	5.7		5.7		
	0.5	5.0		5.0		

TABLE 5

Effect of diet on the neutralizing action of saliva (O. O.; susceptible to caries)

DATE	HCl ADDED cc.	TIME OF COLLECTION 11:45 pH	11:45 P: mg. per 100 cc.	12:45 pH	12:45 P: mg. per 100 cc.	REMARKS
Oct. 30	0	6.7		6.8		Regular diet
	1.0	5.6		5.8		
	0.5	4.9		4.7		
Nov. 4	0	6.8		6.6		Diet as above
	1.0	5.7		5.6		
	0.5	5.0		4.8		
" 6	0	6.8	8.6			Diet as above
	1.0	5.4				
	0.5	4.0				
" 10	0	6.4	9.3	6.8		Diet 1, started Nov. 7
	1.0	4.8		5.3		
	0.5	4.2		4.7		
" 13	0	6.4	17.1	6.8	15.4	Diet as above
	1.0	5.4		5.5		
	0.5	4.9		5.1		
" 18	0	6.7	12.0	6.8	15.7	Diet 3, started Nov. 14; apples taken primarily for fruit
	1.0	5.3		6.0		
	0.5	4.5		5.0		
" 24	0	6.7	18.4	6.8	13.8	Diet as above
	1.0	5.8		5.8		
	0.5	5.3		5.3		
" 25	0	6.5	18.0			Diet as above
	1.0	5.7				
	0.5	5.1				
Dec. 2	0	6.9		6.9		Diet as above
	1.0	6.1		6.1		
	0.5	5.3		5.3		
" 4	0	6.7	18.1	6.8	18.0	Two bananas daily started as fruit on Dec. 3
	1.0	5.9		5.8		
	0.5	5.0		5.1		
" 8	0	6.9	15.4	6.9	14.7	Diet as above
	1.0	5.9		6.1		
	0.5	5.3		5.4		
" 9	0			7.0	15.1	Diet as above
	1.0			6.3		
	0.5			5.6		
" 11	0	6.9	16.2	6.9	18.2	Diet as above
	1.0	5.7		5.7		
	0.5	5.1		5.2		
" 16	0	6.9	15.0	6.8	16.6	Diet as above
	1.0	5.7		5.8		
	0.5	5.0		5.2		

TABLE 6

Effect of diet on the neutralizing action of saliva (T. G. F.; decay rampant)*

DATE	HCl ADDED cc.	pH	P: mg. PER 100 cc.	REMARKS
Oct. 23	0	6.7		Regular diet including milk, meat, eggs, vegetables, fruit
	1.0	5.2		
	0.5	4.5		
" 30	0	6.4		Diet as above
	1.0	4.9		
	0.5	4.4		
" 31	0	6.4	16.1	Diet as above
	1.0	5.2		
	0.5	4.5		
Nov. 10	0	6.5	12.9	Fruit intake increased to at least three oranges a day, beginning Nov. 7
	1.0	4.9		
	0.5	4.3		
" 11	0	6.4	11.7	Diet as above
	1.0	4.4		
	—	—		
" 15	0	6.5	15.9	Diet as above
	1.0	5.0		
	0.5	4.3		
" 17	0	6.3	19.7	Diet as above
	1.0	4.8		
	0.5	4.4		
" 18	0	6.5	15.1	Diet as above
	1.0	5.3		
	0.5	4.6		
" 20	0	6.2	13.0	Diet as above
	1.0	4.8		
	0.5	4.2		
" 24	0	6.0	12.3	Diet as above; eggs increased; bananas instead of oranges, beginning Nov. 24
	1.0	4.4		
Nov. 30	0	6.5		Diet as above
	1.0	5.0		
	0.5	4.4		
Dec. 4	0	6.1	17.3	Diet as above
	1.0	5.2		
	0.5	4.6		
" 8	0	6.5	12.5	Diet as above
	1.0	5.2		
	0.5	4.5		
" 11	0	6.4	9.2	Diet as above
	1.0	5.5		
	0.5	4.8		

* All samples were collected from 12 to 1 o'clock.

Diet 1—cream of wheat, oatmeal, puffed rice, white bread, white of egg, gelatine, butter, cream, and tea or coffee. *Diet 2*—large amounts of fruit, vegetables, and milk, with only moderate amounts of meat, and very little bread. *Diet 3*—similar to Diet 2, but fruits and vegetables were somewhat reduced, and meat supplemented with eggs considerably increased. Subject *T. G. F.* (*table 6*) was kept on his regular diet of moderate amounts of milk, meat, eggs, vegetables, bread and fruit, except that the fruit intake was markedly increased. He had very active tooth decay. As he was quite subject to colds[1] and to sinus infections, his diet was routinely supplemented with cod-liver oil.

Discussion of results. The recorded results show that variations in diet produced changes in the salivary acid-neutralizing power in all except one (*table 6*). The results are in agreement with those obtained by Hawkins (2), who showed that the pH of saliva (as measured about one hour after collection), which he assumes indicates acid-neutralizing power, is lowered by a high-cereal diet or a high-grain diet, but increased by a high-vegetable-and-fruit diet containing moderate amounts of meat and sufficient calcium. He found that acute or pronounced chronic infections, pregnancy, and regular use of magnesium salts also tended to lower salivary neutralizing-power as measured. It is possible that the non-response obtained with subject *T. G. F.* in these experiments is due to his sinus infection, or to some other unknown chronic infection. Similarly the effect of a severe cold, as shown by subject *C. P.* (*table 2*), further emphasizes the marked effect of certain infections on salivary reaction. Subject *R. J.* also developed a moderate cold during the course of the experiment, but in this case no apparent change in his saliva was noticed. The response obtained in the case of subject *R. C. M.* (*table 3*), when placed on Diet 1, was slight, until the collection period of November 20. The marked drop obtained on that day can hardly be due to any metabolic disturbances preceding the headache and fever of indeterminate origin starting on November 23, since on November 25 a marked increase in neutralizing action was evident, although the subject was still feverish, and the night before had a temperature of

[1] We use "cold" conveniently throughout to signify "head cold."

102°F. Examination of the diets for inorganic-salt content would show that Diet 1 is low in potassium, phosphorus, and calcium, and also potentially acidic. The decrease in salivary neutralizing-power correlated with this diet is presumably not due to its potential acidity but to its low content of inorganic salts. This is indicated by the fact that, for subject *J. C. F.* (*table 1*), the salivary neutralizing-power was just as high on a diet of bread, cereals, and meat as on a diet high in vegetables, meat, and fruit; for subject *C. P.* (*table 2*), the low neutralizing-power on Diet 1 was rapidly raised to normal by the addition of a small amount of meat and milk. Examination of the phosphorus values shows no correlation between neutralizing power and phosphate concentration of saliva. This is in agreement with the results obtained by Karshan and co-workers (3), and those to be reported by one of the authors (1).

Although from the limited number of cases reported it is impossible to state that there is a constant relationship between salivary acid-neutralizing power and dental caries, it is interesting to note that the subject immune to caries had a very high salivary acid-neutralizing action, while the very susceptible one had the lowest acid-neutralizing action. The values obtained for the others were in between. However subjects *J. C. F.* and *C. P.*, who at present are relatively immune to caries, showed a normal acid-neutralizing power greater than subjects *O. O.* and *R. J.*, who are quite susceptible. Further investigation in this direction is in progress.

Summary. A high-cereal or a high-grain diet tends to decrease salivary acid-neutralizing power, while a high-meat, high-egg, and high-vegetable-and-fruit diet tends to increase it. No relation was found between the acid-neutralizing power and the phosphorus concentration of saliva. The elements in food may be more important than its potential alkalinity in determining salivary-neutralizing power.

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STUDY OF THE HUMAN FOETAL MANDIBLE

I. Nature of Its Change of Shape

THOMAS R. INGHAM, S.B.

Department of Dental Research, Harvard University Dental School, Boston, Mass.

Introduction. Perhaps the most comprehensive of recent articles on the development of the foetal mandible are those by Low (1) and Wissmer (2). The latter presents a particularly able summary of the enormous amount of work on this subject. One aspect, which on the whole has been little studied, is the change of shape of the foetal mandible in the course of its growth. The present paper indicates the nature of the shape-change. Man, though amazingly ever the same in structural design, yet varies so greatly in individual details that a determination of the common points of the details requires study of many normals. This general law applies directly to this work, in which we obtained measurements on foetuses which, so far as can be determined, represent normal human skeletons at different prepartum ages. Obviously these foetal frames are not likely to be entirely characteristic of the normal, for their existence in a museum indicates abnormality either in mother or foetus, or both. Therefore, our conclusions, although stated positively, may not apply to any other group. Until many such observations have been recorded, general principles can not be established. The foetal skeletons were obtained in the Warren Museum (3). Their growth ages were estimated by referring crown-rump dimensions to Streeter's tables (4). All the measurements in *table 1* are in centimeters, and were made with small calipers, a millimeter rule, and a hand lens. This method is not nearly so accurate as Wissmer's (2) examination with a dissecting microscope, but gives results sufficiently exact for our purpose.

Discussion. Graph 1 indicates the ratio of mandibular-arch width at the angles to jaw-width at coronoid process. The plotted data form a smooth curve having its greatest slope-change in the early months of foetal life. The ratio of these lateral dimensions indicates

that, as the foetal jaw matures, the alveolar plate becomes relatively wider, compared with skull width (from 60 percent in early foetal life to 78 percent in late). *Graph 2* shows the ratio of length of alveolar plate, or body of mandible, compared with length of ramus. The

TABLE 1

Data on the age and measurements (cm.) of human foetuses

FOETUS NUMBER	AGE	CROWN-RUMP	TOTAL LENGTH	OCCIPUT-SACRUM	TOTAL WIDTH	ANGLE[1] WIDTH	RAMUS LENGTH	BODY LENGTH	BODY WIDTH
	weeks								
1	28.5	25.3	39.8		5.0	3.4	1.7	3.1	0.8
2	26.5	23.3	38.3		5.0	3.6	1.9	3.0	1.0
3	28.5	25.3	37.8	12.5	4.7	3.2	1.5	2.7	0.7
4	24.5	21.2	35.4	13.4	5.0	3.6	1.7	3.0	0.85
5	21.0	17.5	30.0	11.2	4.1	2.6	1.6	2.3	0.65
6	21.5	18.0	29.5	11.0	3.9	2.7	1.6	2.3	0.55
7	19.0	15.2	24.5	10.2	3.1	2.0	1.1	1.9	0.45
8	16.0	11.6	19.8	6.6	3.0	1.7	1.0	1.8	0.35
9	17.0	13.5	22.9	9.7	3.1	2.4	1.2	1.9	0.45
10	16.0	11.7	20.2	7.0	3.1	2.1	1.1	1.9	0.35
11	15.0	9.5	15.5	5.5	2.3	1.5	0.9	1.5	0.35
12	15.3	10.6	16.3	6.5	2.5	1.7	0.8	1.4	0.33
13	15.5	10.8	17.6	6.9	2.4	1.8	0.8	1.5	0.35
14	14.5	9.0	13.5	5.7	1.6	1.0	0.7	1.0	0.28
15	12.5	6.5	9.5	4.0	1.3	1.0	0.5	1.0	0.23
16	10.8	4.6	7.0	3.0	0.7	0.4	0.3	0.65	0.18
(From the Wm. R. Lawrence Collection of Skulls, in the Warren Museum)[2]									
17	12.5				2.1	1.5	0.6	1.4	
18	25.0				4.2	3.0	1.3	2.6	
19	34.0				5.5	4.3	1.8	3.6	
20	38.0				5.8	4.7	1.8	3.8	

[1] Angle width: the distance between the two angles of the lower jaw.

[2] The ages of specimens Nos. 17 to 20, inclusive, were estimated from clinical history. No. 17 seems older than the age specified.

growth rates are such as to keep this ratio nearly constant during the greater part of foetal life. Computed in terms of alveolar-plate length to total jaw-length (alveolar plate plus ramus), this ratio is about 63–64 percent. In the early and late parts of foetal life, the alveolar plate comprises a greater part of the mandible length (67

percent in the eleventh week and again in the fortieth), but this may be an erroneous observation owing to lack of sufficient material. Wissmer (2) similarly considers the growth ratios, but for compara-

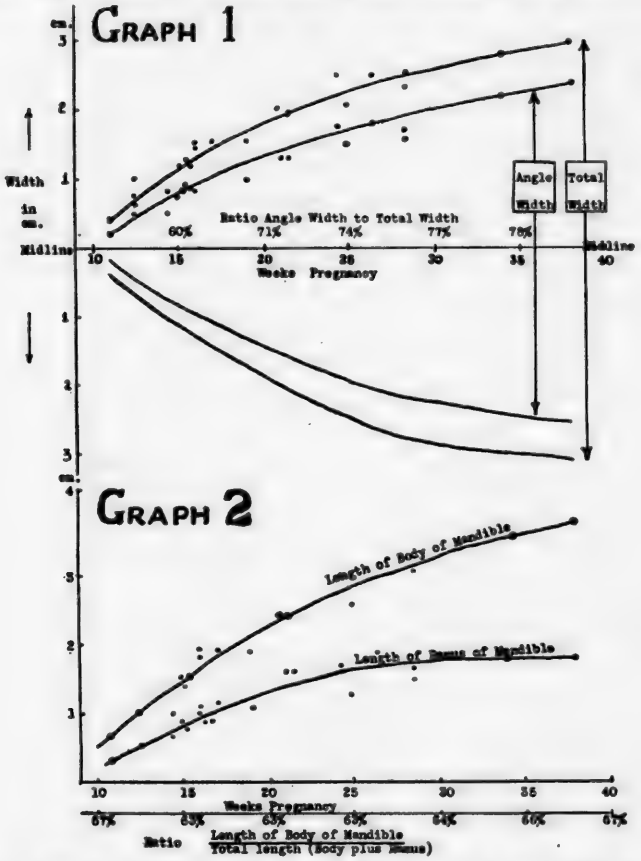

FIG. 1. *Graph 1:* ratio of mandibular-arch width at angles to jaw—width at coronoid process. *Graph 2:* ratio of length of alveolar plate, or body of mandible, compared with length of ramus.

tive measurements chooses a different alveolar-plate length—the distance from mental foramen to symphysis. From a study of thirteen cases he determined the following ratios between alveolar plate and total jaw-length.

Age of foetus in months	3	6	7	9
Ratio in percent	24	27	25	33

From this he emphasizes the fluctuation in ratio during the seventh month, the alveolar plate growing relatively more rapidly at first, then less, then more, than the ramus during the course of foetal-jaw development. We believe that both Wissmer's and our specimens are too few in number to justify definite conclusions. Until clearly proved otherwise, it appears reasonable to accept our graphs showing a growth rate that maintains a pretty even ratio between alveolar-plate length and mandible length. We did not compare depth proportions (antero-posterior measurements), since these involve two variables (widening of angle and change in length of parts), which are above considered separately.

Body growth in general is subject to so many variations that many more such measurements must be taken before definite conclusions can be well-grounded. Yamazaki's (5) studies of the symmetry of the foetal jaw show that the quantities above considered—maximum length of ramus and length of alveolar plate—are the most variable jaw measurements. Unfortunately his data on foetal jaws do not indicate age of development, and therefore cannot be directly applied to this problem, i.e., the nature of jaw changes in foetal development.

Summary. From the above data and graphs we conclude that the alveolar plate lengthens more rapidly than the ramus. The ratio of alveolar-plate length to the total mandible-length is relatively constant. The width of alveolar plate increases more rapidly than the total width. The ratio of width between the mandible angle and the total width approaches a constant during the greater part of foetal life.

References to literature, etc.: (1) Low, A.: *Journal of Anatomy and Physiology*, 1910, *44*, 83. (2) Wissmer, A.: *Archiv. d'Anat., d'Hist. et d'Embryologie*, 1927, *7*, 334–425. (3) Warren Museum, Harvard Medical School. For the privilege of examining the carefully selected series of normal skeletons graduated by age, we are indebted to Dr. M. M. Canavan, Curator of the Museum. (4) Streeter, G. L.: Carnegie Institute of Washington, Publication No. 274; Contributions to Embryology, *11*, 143–168. *Head Modulus* (p. 150): expressed in mm.—mean between greatest horizontal circumference of head and biauricular transverse arc. Measurements are conveniently done with strips of paraffined paper. (This method would have applied nicely to Yamazaki's data.) (5) Yamazaki, K.: *Arch. d'Anat., d'Hist. et d'Embryologie*, 1930, *12*, 109–124.

Quotations on the Status of Dentistry (continued)

training of better qualified dentists. Dentistry is a profession which has become firmly established in its own right, and one which comprises a number of specialties. *It would lose much by becoming a minor subdivision of medicine* [italic not in the original]. It had best retain its independence.—*W. McKim Marriott, M.D., Dean and Professor of Pediatrics, Medical School, Washington University, St. Louis, Mo.; Journal of Dental Research*, 1931, xi, p. 807; October.

Medicine and medical education have troubles of their own which will not be alleviated by attempting to swallow whole another profession; a profession, which, by and large, does not want to be engulfed. And a profession like dentistry, with a hundred-year history, deserves to have its feelings considered. You may call dentistry a specialty of medicine, but that does not make it so; and it is none the less valuable for that fact. I can see no reason why the degree, Doctor of Dental Surgery, honestly held and honorably upheld, should not in the future deserve equal respect and esteem from the public with the degree of Doctor of Medicine, provided it is held by a similar type of man. And whatever their future relationship may be, let medicine and dentistry now go forward side by side as friendly collaborators in the estimable undertaking of offering help to those who need it and adding to the happiness of life as much as to the length of its span.—*F. T. Van Beuren, Jr., A.B., M.D., Associate Dean, and Associate Professor of Surgery, Columbia University College of Physicians and Surgeons, New York City; Journal of Dental Research*, 1932, xii, p. 249; April.

We believe that the best interest of the dental profession, the public and all others concerned can best be served by a *continuation* of the separate organization of the dental profession. The fundamental soundness of this contention has been proved by the ninety-two years of continuous and remarkable progress of the dental profession, despite many handicaps, and important and serious opposition. We are convinced that the dismemberment of the dental profession through adoption of some of its parts by the medical profession, and the relegation of the remainder to comparatively uneducated technicians would seriously depreciate the quality of dental ministrations to the public. We believe that any advantages that might possibly arise from such destruction of the dental profession would be more than equalled by the development of dentistry into the full health service *equivalent* of an oral specialty of the practice of medicine, and this could be accomplished without the disadvantages that would accompany the former procedure.... The fact that the dental profession [like every other profession] falls short of perfection should not subject it to unreasonable attack, especially from those who should be most interested in fostering its progress.—*Report of Committee on Dental Education, New York Academy of Dentistry; adopted by the Board of Governors, April 14, 1931; History of the New York Academy of Dentistry, B. B. Palmer; 1932, p. 51; April.*

Dentistry can best serve the public and promote its own professional attainments by continuing as a separate profession.—*Report of Committee on Dental Education, Minnesota State Dental Association; adopted by the Association; Journal of the Minnesota State Dental Association, 1932, xi, p. 49; April.*

Emphatically, dentistry is a branch and not a mere twig growing on that of medicine, and I contend that the attempt to make it so is responsible for the miserably low standard of dentistry in this country [England]. The dental profession is.... lacking in self-reliance after all these years of subservience and dependence, and is like a flock of bewildered sheep, harassed on every side by conflicting interests..... America the world over holds the premier position in dentistry..... Can you think of a single article of modern surgery equipment which did not originate in the States, and where do you have to go if you want the very best?.... Why is it that in dental affairs England is so far behind America? It seems to me that the reason lies in the control of dentistry by the medical profession..... To adopt a railroad simile, dentistry [in England] has got on to the wrong line. Instead of a dental train controlled by a dental staff, running on its own lines, the dental profession is relegated to a van at the tail-end of the medical train, with practically no choice as to its destination.—*H. C. Visick, L.D.S., R.C.S. Eng.: Journal of Dental Research*, 1932, xii, p. 585; Aug.

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